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CONTENTS OF PREVIOUS NUMBER

MARCH, 1929. NUMBER 3

The Relationship Between Inflammation of the Posterior Sinuses and Disease of the Nervus Opticus. Georges Weill, M.D., Strasbourg, France.

Retinal Degeneration in Macular Region Without Cerebral Symptoms. Arnold Knapp, M.D., New York.

Marked Epiblepharon with Retroflection of the Eyelashes of Both Lower Lids in an Adult Mongolian. Arnold Pillat, M.D., Peking, China.

The Preretinal Artery: An Anatomic Study. Isadore Goldstein, M.D., and David Wexler, M.D., New York.

Cataract in Myotonic Dystrophy. S. R. Gifford, M.D.; A. E. Bennett, M.D., and Nora M. Fairchild, M.D., Omaha.

Ocular Disturbances in Epidemic Encephalitis. Foster Kennedy, M.D., F.R.S. (Edin.), New York.

Circumcorneal Transplantation of Buccal Mucous Membrane as a Curative Measure in Diseases of the Eye. Rudolf Denig, M.D., New York.

Cultivation of Lens Epithelium in Vitro: A Further Study. Daniel B. Kirby, M.D.; Keith Estey, and Florence Tabor, New York.

Obituary:
J. M. Banister, M.D.

Notes and News.

Abstracts from Current Literature.

Society Transactions:
The German Ophthalmological Society.
Société de Neurologie.
Royal Society of Medicine, London. Section on Ophthalmology.

Book Reviews.

Directory.

ARCHIVES of OPHTHALMOLOGY

Volume 1 (old series Volume 58) APRIL, 1929 Number 4

THE FORMATION OF THE AQUEOUS HUMOR

ITS RELATION TO INTRA-OCULAR AND VASCULAR PRESSURES *

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NEW HAVEN, CONN.

More than ordinary importance should be attached to accurate information regarding the composition of the intra-ocular fluid, because such knowledge must be depended on to furnish a clue regarding the origin and alterations thereof. The much debated question whether the aqueous humor represents a specific secretion by a special structure such as the ciliary body and its processes, or whether it is merely a fluid having its origin through relatively simple physical and physicochemical processes from the circulating fluids of the body—the blood and the lymph—hinges on a comparison of the composition of the various fluids involved and a consideration of the forces by which the ocular fluid is produced.

Some of the more important components, such as the sugar, the protein and the chloride, have been studied by several investigators, but the other constituents have scarcely been touched on. No one species of animal has been utilized consistently; hence, a compilation of comparative results has been almost impossible. This is not the place in which to present a detailed critique of the shortcomings of the earlier experiments. The results show a lack of agreement with respect to the quantitative occurrence of most of the constituents. Many of the data involved have recently been summarized by Duke-Elder [1] and others.

It has been my purpose to determine with analytic precision the composition of the normal intra-ocular fluid of living animals and to compare the results, when possible, with those for their blood and spinal fluid. All the analyses were made on single specimens of the aqueous humor withdrawn from the anterior chamber of the eye. I was confronted with the difficulty that other workers have been obliged to face, namely, the small quantity and the minute concentration of some of

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* From the Section of Ophthalmology, Department of Surgery, Yale University School of Medicine.

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* Read before the American Academy of Ophthalmology and Otolaryngology at St. Louis, Oct. 15, 1928.

1. Duke-Elder, W. S.: Recent Advances in Ophthalmology, Philadelphia, P. Blakiston's Son & Company, 1927; The Nature of the Intra-Ocular Fluids, Brit. J. Ophth. (Monograph Supplement III), 1927.

the substances in the aqueous humor. The average values that I secured in the laboratory for the constituents of this fluid, the spinal fluid and the serum of the dog are summarized in the accompanying table. These values compare favorably with those determined by others on the same species.

In the results of the analyses recorded in the table, it may be noted that, so far as all the major constituents are concerned, the composition of the aqueous humor in the dog has a striking resemblance to that of the serum and that of the cerebrospinal fluid. The chemical composition of the ocular fluid appears to be nearly identical with that of the cerebrospinal fluid. The aqueous humor reveals a small amount of protein as compared with the serum. The concentration of sodium chloride, on the other hand, is considerably higher in the aqueous humor than in the blood serum.

Comparison of Quantitative Determinations of the Composition of Intra-Ocular Fluid, Spinal Fluid and Serum of the Dog

Components	Milligrams per 100 Cc. of Fluid		
	Intra-Ocular Fluid	Spinal Fluid	Serum
Dextrose	91.9	85.1	79.1
Sodium chloride	622	508	497
Total nitrogen	35.2	30.4	....
Nonprotein nitrogen	28.6	29.1	33.8
Protein nitrogen	6.6	7.5	....
Amino-acid nitrogen	9.1	....	....
Urea nitrogen	12.1	....	....
Uric acid	1.7	1.7	1.7
Inorganic sulphate (determined as SO_4)	0.36	....	....
Inorganic phosphate (determined as P_2O_5)	1.1	....	....
Refractive index	1.33516	1.33512	
Carbon dioxide per cent by volume per 100 cc	61.7	52	46.3

Lehmann and Meesmann,[2] Duke-Elder,[1] Cohen, Killian and Metzger [3] and Wittgenstein and Gaedertz [4] showed that the concentrations of sodium, potassium, calcium and magnesium ions in the aqueous humor are less than in the blood serum, and that the concentrations of chloride, phosphorus and sulphur ions are greater in the aqueous humor than in the serum. It appears, therefore, that there is a difference in the ionized constituents of the fluids. The cations are present in less concentration and the anions in greater concentration in the aqueous humor than in the serum. The remaining substances found in the serum are distributed almost equally in both fluids. The constituents of the fluids may be divided into two groups, according to the charactertistics of the mole-

2. Lehmann and Meesmann: Arch. f. d. ges. Physiol. **205**:210, 1924.

3. Cohen, M; Killian, J. A., and Metzger, M.: Contributions to Ophthalmic Science, Jackson Birthday Volume, Menasha, Wis., George Banta Publishing Company, 1926, p. 216.

4. Wittgenstein, A., and Gaedertz, A.: Arch. f. Ophth. **119**:395, 403 and 755, 1927.

cules in solution. To the first group of slightly diffusible compounds belong such ingredients as the proteins; to the second group belong the readily diffusible substances that are distributed in the plasma and the aqueous humor. The latter group may again be divided into non-dissociated and dissociated diffusible substances.

Mestrezat and Magitot,[5] Van der Hoeve [6] and Osborne,[7] employing the method of depression of the freezing point, found the aqueous humor and the serum essentially isotonic; and Duke-Elder,[1] by means of a micro-osmometer, noted that the isotonicity corresponds to that of a dialysate of the blood in the arterial capillaries. The specific gravity of the aqueous humor of the dog has been determined as varying between 1.007 and 1.009 and the refractive index as varying between 1.3335 and 1.3360. The surface tension estimated by Dieter [8] was from 72.4 to 73 dynes per centimeter at 18 C. The viscosity of the aqueous humor in the dog was found by Scalinci [9] to be greater than that of water and less than that of blood. The conductivity of the aqueous humor was found to be greater than that of serum. Van der Hoeve [6] found the conductivity to be the mean between the dialysate of arterial and that of venous blood. The hydrogen ion concentration was estimated to be from p_H 7.40 to p_H 7.50, which is slightly higher than that of serum. It appears, therefore, that the normal aqueous humor is a product of the blood and is separated from it through a membrane that is relatively impermeable to molecules of large size. Those constituents which are ionized are partitioned variably in the two fluids, whereas the nonionized diffusible substances are found in equivalent proportions.

After the foregoing consideration of the composition and physical properties of the aqueous humor and its comparison in these respects with other fluids of the body, a presentation of the evidence supporting the various hypotheses may lead to a better understanding of the probable process of the formation of the intra-ocular fluid.

SECRETION THEORY

The supporters of this theory have considered the ciliary body and its processes as the chief source of the intra-ocular fluid. The functions of these tissues of the eye have been likened to that of the choroid plexus of the cerebrospinal system because of their anatomic structure and cellular performance. The ciliary body and its processes were regarded as a gland by early observers. Nicati [10] and others described the epithe-

5. Mestrezat, W., and Magitot, A.: Compt. rend. Soc. de biol. **84**:185, 1921.
6. Van der Hoeve, J.: Arch. f. Ophth. **82**:58, 1912.
7. Osborne, W. A.: J. Physiol. **52**:346, 1918-1919.
8. Dieter, W.: Arch. f. Augenh. **96**:8, 1925.
9. Scalinci, N.: Arch. f. Augenh. **57**:214, 1907.
10. Nicati: Arch. d'opht. **11**:24, 1891.

lial layer of these structures as glandular in nature. Boucheron [11] considered the anterior part of this area as secreting the aqueous humor, and the posterior part as secreting the vitreous. Later, Collins [12] described an epithelial outgrowth covering the ciliary body and its processes as ciliary glands. These observations, however, are not in accord with the histologic studies presented more recently by Finnoff,[13] Salzmann [14] and others.

Seidel and Schmelzer [15] observed cytologic changes in the epithelial covering of the ciliary processes following cellular activity, which were considered by them as manifestations of glandular processes. They found an increase in the size of the cell, which also differentiated itself into two zones: a granular area at the inner border and a clear zone at the periphery or free margin of the cell. Hemosiderin granules and mitochondria were also observed in the tissues. The same cellular disturbance was noted in the choroid plexus under similar conditions. Becht [16] pointed out that, in the latter, these changes in the cells did not constitute evidence of active secretion, and the alteration of cellular tissue was the exact opposite of that which occurs during active secretion in typical glands. Weed [17] also demonstrated that the intravenous injection of distilled water caused analogous changes in the choroid plexus. He interpreted the alteration in the cells as evidence of an increased amount of fluid passing through them.

Further evidence of active secretion was sought by Nicati,[10] Seidel and Schmelzer [15] and others by introducing into the general circulation, and locally into the culdesac, such agents as pilocarpine and physostigmine, which produce true secretion in other glands of the body. Changes in cellular tissue were noted as described, and the manifestation was accompanied by an increased content of protein in the aqueous humor. The reactions to the miotics were in accord with those set forth by Wessely,[18] and by my co-workers and me,[19] but this result cannot be

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11. Boucheron: Bull. et mém. Soc. franç. d'opht. **1**:81, 1883.

12. Collins, E. T.: Tr. Ophth. Soc. U. Kingdom **11**:53, 1891.

13. Finnoff, W. C.: Arch. Ophth. **44**:626, 1915.

14. Salzmann, M.: Anatomy and Histology of the Human Eyeball, Translated by E. V. L. Brown, Chicago, University of Chicago Press, 1912.

15. Seidel, E., and Schmelzer, in Abderhalden, Emil: Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwartzenberg, 1927, vol. 5, p. 969.

16. Becht, F. C.: Am. J. Physiol. **51**:1, 1920.

17. Weed, L. H.: Am. J. Anat. **32**:253, 1923.

18. Wessely, K.: Centralbl. f. prakt. Augenh. **37**:303, 1918.

19. Yudkin, A. M.: Aqueous Humor; Critical and Experimental Study, J. A. M. A. **87**:1910 (Dec. 4) 1926. Yudkin, A. M.; Krause, A. C., and Morton, D. G.: Experimental Transmission of Arsenic to Aqueous Humor, Arch. Ophth. **57**:147 (March) 1928.

interpreted as necessarily indicative of glandular activity. That it was not due to glandular activity is indicated also by the work of Köller [20] and Theil,[21] who produced evidence to show that physostigmine causes a vasodilatation in the ocular tissue; and Adler and Landis [22] demonstrated that even though the ciliary ganglion is severed or degenerated, the miotics produce the same effect and hence the action must be vascular in origin, rather than glandular. It was shown that atropine had the opposite effect on the vascular tissue.

Seidel [15] further supported the theory that the intra-ocular fluid is a secretion by showing that there is a difference of electrical potential between the ciliary body and its processes and the cornea, so that a current can be established, as in the case of a secretory structure such as the salivary glands; but this manifestation was questioned by Lullies and Gulkowitsch [23] and Duke-Elder.[1] They believed that this electrical phenomenon might be explained by the fact that the two electrodes that Seidel [15] used were immersed in solutions of different ionic concentration.

It is a known fact that in the active secretion of a gland there is usually some substance newly formed within the cell for some specific purpose in the body. For example, the salivary and pancreatic secretions contain enzymes not found in the blood, which are necessarily produced de novo within the glandular structures. So far as I know, substances not occurring in the blood have not been detected in the intra-ocular fluid.

In view of the fact that many authorities question whether the action of the miotics, pilocarpine and physostigmine, and the cellular changes produced by them and other means are evidence of true secretion, and in view of the fact that the ciliary body and its processes do not resemble other glandular tissue histologically, it is necessary to seek an explanation of the composition of the intra-ocular fluid in some other method than that of true glandular secretion.

FILTRATION THEORY

In opposition to the theory that the intra-ocular fluid is a physiologic secretion is the theory that it is formed by filtration, advocated by Leber [24] and his school. They believed that the intra-ocular fluid is produced in the ciliary processes by physical filtration, the principal factor

20. Köller: Arch. Ophth. **50**:550, 1921.

21. Theil, R.: Arch. f. Ophth. **113**:347, 1924.

22. Adler, H. F., and Landis, E. M.: Studies on Protein Content of Aqueous Humor; Effects of Miotic and Mydriatics, Arch. Ophth. **54**:265 (May) 1925.

23. Lullies, H., and Gulkowitsch, L.: Zentralbl. f. d. ges. Ophth. **13**:463, 1924.

24. Leber, T., in Graefe Saemish: Handbuch der Gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1903, vol. 2, p. 207.

in this performance being the gradient pressure from the point of origin to the point of absorption. This assumption was supported, in the main, by the experimental work of Henderson and Starling,[25] who showed that the production of intra-ocular fluid is strictly proportional to the difference of pressure between the blood in the capillaries of the eyeball and the intra-ocular fluid. They believed that the difference of pressure, amounting to over 30 mm. of mercury, is sufficient to account for the low content of protein in the intra-ocular fluid without the assumption of an active intervention on the part of the capillary walls or of the ciliary body.

Knowledge of the factors involved in exchange of fluid has been considerably modified and augmented during recent years. All experimental work on this subject, however, reveals that the capillary membrane ordinarily is not permeable to the proteins of the blood plasma which circulate through its lumen, and that the permeability of the membrane varies in different species of animals and in different parts of the body in the same individual. It has been shown that the proteins of the plasma, unable to leave the capillaries, produce an osmotic pressure that tends to retain the fluid in the blood vessels, and that the capillary blood pressure acts in the reverse manner. Krogh[26] demonstrated that the capillaries constrict and dilate independently of the arterioles, and that their caliber, when the tissue they supply is at rest, may be much less than when the tissue is active. This idea is not in accord with the older belief that, with the heart functioning normally, the extent of the capillary circulation depends primarily on the contraction and dilatation of the arterioles and secondarily on the venous pressure. Recently, Landis,[27] utilizing a delicate experimental technic, showed that the dilated capillaries are not more permeable than the constricted vessels, and that an increase in capillary pressure would cause water to pass toward the tissues, while the converse relation would lead to the movement of fluid into the blood. He also demonstrated that an injury of the capillary endothelium increases its permeability to protein, with a corresponding reduction of the colloid osmotic pressure. He believed, therefore, that the direction and the amount of the flow of water through the normal capillary wall are determined primarily by the level of capillary pressure in association with osmotic forces.

Although the present knowledge of capillary permeability may account for some of the observations on the intra-ocular fluid, further explanation is necessary in view of the more recent contributions con-

25. Henderson, E. E., and Starling, E. H.: Proc. Roy. Soc., London **77**:284, 1906.

26. Krogh, A.: The Anatomy and Physiology of the Capillaries, New Haven, Conn., Yale University Press, 1922.

27. Landis, E. M.: Am. J. Physiol. **81**:124, 1927.

cerning the chemical content and the physical properties of the aqueous humor. It was noted in a previous paragraph that the aqueous humor possesses considerably less protein than the serum, that its content of sodium chloride is greater than that of serum, that the undissociated diffusible crystalloids, which are largely represented by dextrose, appear to be present in both systems in apparently equal concentration and that, of the dissociated diffusible crystalloids, the cations sodium, potassium, and calcium magnesium are in less concentration, and the anions chlorine, phosphate and sulphate in greater concentration in the aqueous humor than in the serum.

A critical review reveals that Mestrezat and Magitot [5] observed a parallel difference in the concentrations of individual substances in the cerebrospinal fluid and the blood serum and that the osmotic pressures of the two fluids are approximately isotonic, when measured by the depressions of the freezing points. By further investigation, they found that the aqueous humor, like the cerebrospinal fluid, is an equilibrated dialysate of the blood serum. Baurmann and Meesmann [28] observed a similar interchange of the chemical constituents in the blood serum and the aqueous humor. Fremont-Smith [29] also noted this physicochemical arrangement. He was able to show that a relation exists between the concentration of proteins in the plasma and the distribution of chlorides, so that the greater the content of protein in the plasma, the greater the excess of chlorides in the cerebrospinal fluid. He found that an analogous condition prevails between plasma and pleural, and ascitic and synovial effusions, respectively. It therefore appears that the formation of the aqueous humor must be accounted for in some other way than by the process of secretion or of simple filtration by pressure.

Recently, a different view was proposed by Duke-Elder,[1] Lehmann and Meesmann,[2] Baurmann and Meesmann,[28] Mestrezat and Magitot [5] and others. They believed that the unequal distribution of the ions in the two related fluids, serum and intra-ocular fluid, is characteristic of a system in membrane equilibrium by virtue of their relationship. Duke-Elder [1] championed the membrane equilibrium theory enunciated by Donnan [30] as applicable to the conditions of the formation of the fluid in the eye. Not only does the normal fluid obey this theory, according to Duke-Elder, but the abnormal aqueous humor likewise supports the Donnan theory of membrane equilibrium. He considered the capillary walls and not the ciliary epithelium as the essential dialysing membrane,

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28. Baurmann, M., and Meesmann: The Intraocular Fluids, Klin. Monatsbl. f. Augenh. **74**:518, 1925.

29. Fremont-Smith, F.: The Nature of the Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **17**:317 (March) 1927.

30. Donnan, F. G.: Ztschr. f. Elektrochem. **17**:572, 1911.

because the intra-ocular fluids are in equilibrium with the capillary blood, the properties of the aqueous humor vary directly with the state of their permeability and the ciliary region is not the only site of the process of dialysis. He postulated "that," in such a system, "there is a continuous process of reciprocal interchanges on either side of the capillary walls, made up of filtration outwards, determined by the differences in the hydrostatic pressures, an osmotic flow in the opposite direction, conditioned by the relative osmotic pressures, a diffusion out or in of the diffusible substances according to their several differences in concentration and a readjustment depending on colloid impermeabilities, and that on either side of the capillary walls there is a balancing hydrostatic and osmotic pressure equilibrium around which level the mutually compensating pressures are continually fluctuating, and in any capillary these balancing forces will determine a flow of fluid outward at one movement and inwards at another." Theoretically, the fluid is stagnant, but superimposed on this primary metabolic interchange, there probably is a secondary and intermittent circulation which is determined by the contraction of the internal and external musculature, the influence of the pulse beat and other factors.

COMMENT

From a chemical point of view, my observations of the normal and the abnormal composition of the intra-ocular fluid are in accord with those presented by the advocates of the theory of a membrane equilibrium. This agreement, alone, is not sufficient to warrant acceptance of the hypothesis proposed by Duke-Elder;[1] other factors must be considered also. It has been shown that when a membrane is more permeable to one ion than to another, a variable electrical potential and chemical reaction exists in the separated fluids, and the osmotic and hydrostatic forces are equalized. This has been established by the experimental work of Mestrezat and Magitot [5] and others with colloidal sacs of known permeability. A critical study, however, reveals that the existence of parallel thermodynamic forces between the capillary blood and the intra-ocular fluid in vivo cannot be demonstrated with the present technic. As yet, a direct method, with the exception of the micro-osmometer of Duke-Elder,[1] has not been devised for measuring these forces in the blood and the eye fluid of the same animal under normal physiologic conditions.

There are, however, certain chemical and physiologic changes which take place in the eye when the composition of the blood is altered and the permeability of the capillaries is changed, which point toward the theory of membrane equilibrium. Certain disturbances of the osmotic and hydrostatic pressures of the two systems produce changes that may

be accounted for more readily by the assumption of this hypothesis. A review of these observations may help in understanding the situation better.

Duke-Elder [1] injected large quantities of gum arabic into the blood of cats and found that a change did not occur in the refractive index. Neame and Webster [31] and my co-workers and I [19] demonstrated that arsenic, even though injected intravenously, did not appear in the intra-ocular fluid until the permeability of the vessels was altered. Wittgenstein and Gaedertz [4] showed that intravenously injected diffusible basic dyes and inorganic cations cannot be recovered from the normal aqueous humor under ordinary physiologic conditions. The situation is reversed for diffusible acid dyes and anions. This behavior is attributed to the protoplasmic absorption of the substances. The cathodal substances are absorbed rapidly from the blood stream, and the amount required for transmission of any to the aqueous humor is enormous. In large amounts they are exceptionally toxic. The anodal substances, on the other hand, are absorbed poorly, and hence accumulate to a greater extent in the blood stream, and therefore less is necessary for transmission of some of it to the intra-ocular fluid. From this it is concluded that the diffusible electrolyte depends for its transport on the function of its electrical charge. Van Creveld and de Haan [32] showed that intravenously injected fluorescein was present in the aqueous humor in exactly the same concentration relative to the plasma as they obtained by dialysing blood through collodion membranes. It has likewise been shown that iodide, given orally, subcutaneously, subconjunctivally and intravenously, finds its way into the intra-ocular fluids, and that its concentration in the aqueous humor varies with its concentration in the blood. Bromide and salicylate have also been recovered in the aqueous humor after administration by mouth.

With disturbances of carbohydrate metabolism resulting in either hypoglycemia or hyperglycemia, fairly comparable alterations in the concentration of dextrose in the aqueous humor are exhibited. I have noticed, however, that in either extreme, the content of sugar is greater in the aqueous humor than in the blood. When the concentration of colloidal substances in the blood is altered, transmission of these substances into the intra-ocular fluid does not occur, this failure of transmission probably being due to the large size of their molecules. On the other hand, it seems that diffusible substances aline themselves according to (*a*) their affinity for plasma proteins and (*b*) their ability to dissociate into basic and acid radicles. Nondissociable diffusible ingredients of the blood plasma are partitioned relatively equally on both sides of the membrane.

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31. Neame, H., and Webster, J.: Tr. Ophth. Soc. U. Kingdom **43**:381, 1923.
32. Van Creveld, S., and de Haan, J.: Biochem. Ztschr. **123**:190, 1922.

It has also been shown by Duke-Elder,[1] myself [19] and others that the composition of the intra-ocular fluid is considerably modified when the permeability of the capillary walls is altered (*a*) by a lowering of the intra-ocular pressure, either by a paracentesis of the anterior chamber or by removal of the vitreous humor, (*b*) by local irritation produced by mechanical manipulation or subconjunctival injections of chemicals, (*c*) by stimulation of the cervical sympathetic nerve and (*d*) by radiant energy (Duke-Elder). The product of increased permeability has a greater amount of protein and a decreased concentration of salt, while the nonionized diffusible substances have an apparently equal distribution in both fluids. Duke-Elder [1] also noted that the concentrations of the anions relatively decrease and those of the cations increase with the increase in the concentration of colloids, so that the altered intra-ocular fluid appears to be formed by the same process which produces the normal fluid. From this observation it may be concluded that the intra-ocular fluid is altered by changes (*a*) in the composition of the plasma and (*b*) in the permeability of the capillary wall.

It has likewise been adequately demonstrated clinically and experimentally that the intra-ocular pressure is altered in several ways. By experiment it has been shown that the intra-ocular pressure varies directly with the pressure of the blood. As yet, this parallel relationship between arterial pressure and intra-ocular pressure has not been verified clinically by most ophthalmologists. It has been demonstrated, however, that the intra-ocular pressure varies closely with the venous pressure. Obstruction of the vortex veins as they leave the sclera increases the intra-ocular pressure. Even a ligature on a single vein produces a similar but less marked increase. Carl Ludwig, long ago, pointed out that capillary pressure was far more dependent on venous pressure than on arterial pressure. This observation was confirmed by Carrier and Rehberg [33] in a study of capillary pressure in the hand. The intra-ocular pressure may also be influenced by injections of anisotonic substances into the blood stream. When hypotonic sodium chloride solution is used, the pressure in the eye rises synchronously at first with the blood pressure, and soon drops simultaneously with it. Later, it continues to rise independently of the blood pressure, and remains elevated for a considerable time. On the other hand, a hypertonic solution produces an initial fall in the intra-ocular pressure, which subsequently continues to rise slightly almost parallel with the pressure in the blood. When the injection ceases, the intra-ocular pressure begins to fall without a similar decrease in the blood pressure. The injection of isotonic fluid apparently does not alter the

33. Carrier, E. B., and Rehberg, P. B.: Skandin. Arch. f. Physiol. **44**:20, 1923.

normal status of the intra-ocular pressure. Hertel,[34] Duke-Elder [1] and others showed that the increased intra-ocular pressure of glaucoma may be lowered by hypertonic solutions injected into the blood stream. Corresponding results were obtained experimentally by Fremont-Smith [29] and his co-workers in a study of the cerebrospinal fluid. Clinical corroboration of the lowering of the intracranial pressure was recorded by Cushing [35] and his co-workers.

In order to understand the changes that take place in the intra-ocular pressure, some knowledge of the vascular pressure is necessary. Although several observers have estimated the vascular pressures in the eye, no one as yet has succeeded in presenting a technic that is flawless. The direct method proposed by Duke-Elder [1] appears to have many favorable possibilities but is still experimentally impracticable for all eyes. He calculated the mean pressure in the ophthalmic artery in the cat as about 100 mm. of mercury and that in the retinal artery as about 75 mm. of mercury. He found the pressure in the venous exits in the dog normally slightly above the intra-ocular pressure. This observation of venous pressure is contrary, however, to most experimental reports. From his estimates, he suggested that the capillary pressure in the eye varies from a few millimeters of mercury above the intra-ocular pressure to a height of from 50 to 55 mm. of mercury.

RÉSUMÉ

A recapitulation of the facts shows that the intra-ocular fluid is derived from the blood stream by the same forces that are responsible for other fluids in the tissues. The factors involved in the formation of the fluid are the hydrostatic pressure of the two systems, the permeability of the intra-ocular vascular capillaries and the osmotic pressures of the blood plasma and the intra-ocular fluid. The authorities who believed that the aqueous humor is produced as a pressure filtrate based their assumption on experimental work showing that the hydrostatic pressure in the intra-ocular vascular bed is a gradient one; i.e., the ciliary capillary pressure is greater than the intra-ocular pressure, which in turn is greater than the pressure in the veins and the canal of Schlemm. The composition of the aqueous humor may also be accounted for by the difference between the pressure of the blood stream and that of the intra-ocular fluid. Because of this change in the hydrostatic pressure from the site of origin to the exit of the fluid, a continuous circulation is present. The intra-ocular pressure is considered as a function of the intra-ocular vascular hydrostatic pressure, and it rises and falls with the blood pressure. Some lay stress on the arterial influence and others on that

34. Hertel, E.: Klin. Monatschr. f. Augenh. **66**:924, 1921.
35. Cushing and Foley: Proc. Soc. Exper. Biol. & Med. **17**:217, 1920.

of the venous system. Normal intra-ocular pressure therefore represents the pressure at which the rate of formation of intra-ocular fluid is exactly balanced by the rate of its escape from the eyeball.

The theory that intra-ocular fluid is produced by pressure filtration does not account for the chemical composition of the aqueous humor under either normal or abnormal conditions. If no factors other than filtration are involved in the separation of aqueous humor from the blood, one would expect the composition of the filtrable material to be identical in both fluids. It is known, however, that the proteins are present in the normal aqueous humor only in traces, and that when the permeability of the membrane is altered, their proportions vary according to the laws of diffusion. The diffusible ingredients partition themselves in the fluids according to their electrical potential in the normal and likewise in the altered state. The nondissociable constituents are partitioned almost equally on both sides of the membrane. The physical forces are not so well known as the chemical composition of the fluids, but the changes that present themselves when the hydrostatic and osmotic forces are altered point to the fact that the theory that intra-ocular fluid is produced by pressure filtration needs modification.

CONCLUSION

It is my impression that the intra-ocular fluid is a dialysate of the blood plasma, in the sense that there exists a membrane—the capillary wall and the tissue covering it—which has a functional adaption for keeping the media of the eye transparent and for helping to regulate the slow continuous process of exchange—metabolic and catabolic—on both sides of the membrane, so that there is established a delicate balance between the hydrostatic and the osmotic pressures of the arterial and the venous plasmas within the eye. This balance is the intra-ocular pressure. There is a continuous circulation of the intra-ocular fluid in the eye. The rate of circulation depends on the metabolic exchange and the muscular reaction of the surrounding tissue.

AKINESIS DURING CATARACT EXTRACTION*

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Forced closure of the eyelids, or "squeezing," often with accompanying loss of vitreous, is a frequent and much dreaded complication of cataract extraction. "Squeezing" may be prevented by injections which temporarily paralyze the branches of the facial nerve which supply the muscles of lid closure. Van Lint [1] was the first to publish a method of thus securing akinesis; later, Villard,[2] Wright,[3] Derby [4] and Robinson [5] modified the original procedure. Recently, Wright advocated an entirely different type of injection.

With the exception of Wright in his latest method, all of the surgeons named make their injections at different points near the bony orbital rim. Also, in each case multiple injections are made with a long needle which traverses the tissues for some distance. This type of injection produces some bulging of the soft parts surrounding the lids, which may at times give rise to slight pressure on the globe. In the hands of those who infrequently perform operations, it seems to be difficult to secure akinesis by the methods mentioned.

Wright now recommends injections into the tissues surrounding the facial nerve trunk at its point of emergence from the stylomastoid foramen. Prior to the perfection of the method of making injections into the main trunk, he made a few injections into the region of the parotid gland; however, as he was dissatisfied with the results, he gave up the method. It appears to be difficult to make injections into the main trunk in the region of the stylomastoid foramen and evidently requires considerable practice before one masters the technic. Paralysis of the entire group of muscles supplied by the seventh nerve is produced by this injection. Wright reported the cases of two patients who retained the paralysis over a prolonged period of time, and on whom it was necessary to do a tarsorrhaphy to protect the cornea.

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* Submitted for publication, Dec. 8, 1928.

* From the Department of Ophthalmology, College of Medicine, State University of Iowa.

1. Van Lint: Ophthalmoscope **12**:577, 1914.

2. Villard: Ann. d'ocul. **156**:352, 1920.

3. Wright, R. E.: Am. J. Ophth. **4**:445 (June) 1921; Arch. Ophth. **52**:166 (March) 1923; **55**:555 (Nov.) 1926.

4. Derby, G. S.: Modern Aids to Cataract Extraction, J. A. M. A. **81**:2091 (Dec. 22) 1923.

5. Robinson, J. M.: Arch. Ophth. **53**:570 (Nov.) 1924.

Herewith is presented a method which is recommended because of simplicity of execution and excellence of results. It has been used in more than 200 cases of cataract extraction by various surgeons who operate in the ophthalmic clinic at the University of Iowa. Akinesis of the muscles of lid closure has been complete in almost all cases. The injections were made fairly easily and were practically painless, as an extremely fine, short needle was used. There was no swelling of the tissues in the region of the eye. Usually, only the muscles of lid closure were affected, and no patient has shown evidence of a prolonged paralysis.

As in all surgical procedures, familiarity with the anatomy of the parts involved is necessary. In gentle closure of the eyelids, the orbicularis palpebrarum is the only muscle acting. In forced closure, such as arises as a complication during cataract extraction, other muscles come into play, namely, the frontalis, the corrugator supercilii, the quadratus labii superioris and the pyramidalis nasi.

The entire group of muscles acting in forced lid closure is supplied by the temporofacial division of the seventh cranial nerve. This nerve traverses the upper part of the parotid gland and gives off the temporal, malar and infra-orbital branches. The temporal branches run forward and upward over the zygoma to supply the upper part of the orbicularis, the frontalis, the corrugator supercilii and the pyramidalis nasi muscles. The malar branches run forward over the malar bone to supply the lateral part of the orbicularis and occasionally a part of the quadratus. The infra-orbital branches run horizontally forward to supply the lower part of the orbicularis and the quadratus. Thus, a main branch of the nerve is found which supplies all the muscles of forced lid closure. If one is enabled to locate this temporofacial division and inject into it a sterile 2 per cent solution of procaine hydrochloride, one obtains akinesis of the muscles supplied by it.

The nerve is located in the substance of the extreme upper portion of the parotid gland at a point just external to the condyle of the inferior maxilla. The point of injection is located by palpation in the following manner: The zygoma is located and the under side is followed backward to a point just in front of and slightly below the external auditory meatus. The patient is then asked to open his mouth, and as he does this the condyle of the mandible is felt to slide forward. When the mouth is open, one notes a distinct depression under the finger tip. As the mouth is closed, the condyle is felt to slip back, and it is directly over this point and down onto it that the solution is to be injected. The area of skin over the condyle is touched with tincture of iodine, which serves to mark the point of injection as well as to sterilize the skin. With an ordinary syringe containing a 2 per cent solution of procaine hydrochloride and fitted with a fine sharp needle, the injection is begun

by inserting the needle directly inward until the point strikes bone. The point of the needle is then on the outer surface of the condyle of the inferior maxilla. This point is fairly superficial and is reached quickly. About 1 cc. of the solution is injected, and as the needle is slowly withdrawn, an additional 1 cc. is forced into the tissues.

In edentulous patients, the condyle slips farther backward when the mouth is closed, and one must make note of this; otherwise, the injection is made too far forward. It is necessary to begin the injection with the point of the needle resting against the condyle.

In a large percentage of cases, evidence of oncoming paralysis is noted within from one to two minutes following the injection. The full effect, however, is not evident until about five minutes later.

No ill effects from the injection have been seen. Following operation, care should be exercised to see that the lids are properly closed before the dressing is applied. It is well to use an ointment on the dressing. Usually, dressings are changed on the fourth or fifth day, but some have been renewed after from twenty-four to thirty-six hours, and there has been no sign of prolonged paralysis in any instance.

THE INTRA-OCULAR TENSION FOLLOWING PUNCTURE OF THE ANTERIOR CHAMBER*

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Seidel[1] was the first investigator to find that the intra-ocular tension of the eyes of cats increased after partial or complete emptying of the anterior chamber. Since that time, the eyes of animals have been studied carefully to observe the behavior of the tension after the puncture of the anterior chamber. The tonometer and manometer were used.[2] Attention was drawn to the behavior of human eyes in this respect when Hagen produced his theory of the different modes of regeneration of the aqueous in eyes of human beings and animals. Hagen, himself, reported two cases in 1920; Magitot added a third case. The figures are given in chart 1.

REPORT OF CASES

CASE 1 (Hagen).—A. B., aged 17 years, had a piece of copper in bulbo, resulting in iridocyclitis for six months. The tension was 5.5/10.

CASE 2 (Hagen).—A woman, aged 65, had a small tumor in the region of the posterior pole. The vision was 6/36. There was paracentral scotoma, corresponding to the location of the tumor. The tension was 5.5/10. The anterior chamber, which was of normal depth, was punctured with a syringe. All the aqueous, amounting to about 0.26 cc., was drawn up. The needle was then carried through the anterior chamber and out through the cornea opposite the point of entrance. It was left in this position for the time of the experiment, and the tension was measured every fifteen minutes. The anterior chamber was restored to normal depth four minutes after the puncture.

CASE 3 (Magitot).—A young man had atrophy of the optic nerve resulting from an old cranial injury. The eyeball was entirely normal. The tension in both eyes was 20 mm. of mercury. The anterior chamber of the blind eye was punctured and emptied with little or no suction.

It is easily seen (chart 1) that the three curves are rather different. In case 2, it took ninety minutes for the tension to return to normal; in case 3, the original value was obtained in thirty minutes. The difference must have been due to varying conditions in the eyes examined. In considering this difference, the following questions arise: Which is the curve of the normal eye, and what does this curve indicate?

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* From the Eye Clinic of the University of Chicago, Dr. E. V. L. Brown, Director.

1. Seidel, E.: Arch. f. Ophth. **95**:1, 1918.

2. Seidel (footnote 1); Hagen, S.: Monatsbl. f. Augenh. **64**:187, 1920; ibid. **65**:643, 1920. A. P. Magitot: Arch. Ophth. **52**:1, 1923. Kahn, R. H., and Loewenstein, A.: Arch. f. Ophth. **109**:433, 1922.

The technic used is simple. A fine, sharp needle attached to a 1 cc. syringe is introduced through the cornea near the limbus. The canal should be as long as possible in order to secure absolute tightness after the withdrawal of the needle. For that reason, the needle is introduced obliquely, almost tangent to the surface of the cornea. The aqueous is drawn up until the pupil just begins to contract. Magitot made his punctures in about the same way. Hagen's method has already been described. In order that fluid should not be lost through the hole made with the needle, he left the needle in the eye.

The tiny holes, however, seem to be water tight. The values at which Magitot and I arrived with our simple methods, are higher than Hagen's figures, hence, it is not likely that an appreciable amount of fluid was lost in this technic. Phenacaine was always used for the anesthetic. It is agreed that this drug does not change the circulation of blood in the eye to an appreciable degree.

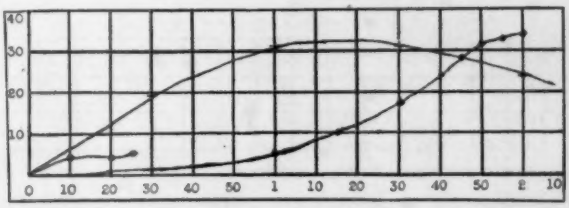

Chart 1.—Tension curve following puncture of the anterior chamber of the eye by Hagen and Magitot. The hollow circles show the change in tension for case 1 (Hagen); the solid circles that for case 2 (Hagen), and the crosses that for case 3 (Magitot). The figures beneath this and the following charts represent the time of the experiment in minutes and hours; those at the left, the tension, measured in millimeters of mercury.

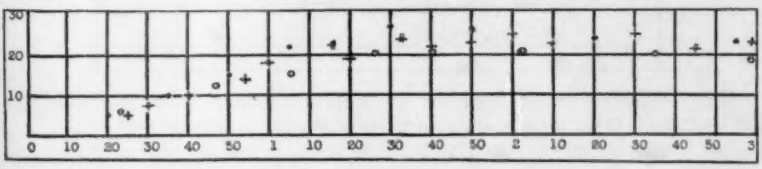

Chart 2.—Changes in tension in four "normal" eyes, after three punctures of the anterior chamber. Each of the symbols, the solid circle, hollow circle, cross and double cross, indicates the tension curve of a single case. The average of these curves is regarded by the author as a standard for comparison.

Chart 2 shows the behavior of four eyes which I think can be considered normal as far as conditions of the circulation of blood and fluid exchange are concerned.

In case 1, the patient had an acute toxic amblyopia due to excessive use of tobacco. He showed small, bilateral central scotomas. He recovered completely after abstinence for three weeks. In case 2, the patient had partial atrophy of the left optic nerve and slight symptoms of adiposogenital dystrophy. Roentgenograms showed a large tumor of the hypophysis. In cases 3 and 4, the patients had strabismus and

amblyopia ex anopsia. These four patients were all under observation in the hospital for at least three days before puncture was performed. All four had an intra-ocular tension within normal or physiologic limits. The daily variations amounted to less than 4 mm. With the slit-lamp the slightest symptom of an inflammatory process could not be detected.

Chart 2 shows that the four eyes behaved similarly after the puncture. The first reading was taken from twenty to thirty minutes after puncture. By this time, the tension was found to be about + 5 mm. of mercury. The original pressure was usually reached in from sixty-five to seventy-five minutes. From this point, the tension kept rising until it exceeded the original value by from 6 to 8 mm. At this point, the summit of the curve was reached, usually in ninety minutes after the

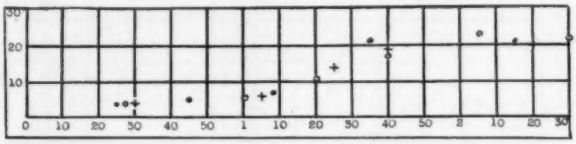

Chart 3.—Tension curves after three punctures, three weeks apart, of the anterior chamber of an eye with tabetic optic atrophy. The curves, as represented by symbols, appear in the following order: the solid circles, the crosses and the hollow circles.

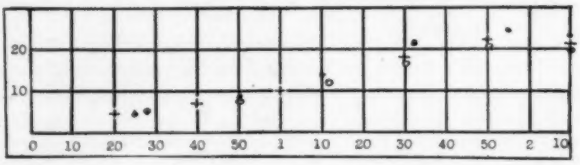

Chart 4.—Result of punctures of the anterior chamber of an eye with tabetic optic atrophy. The solid circles indicate the tension curve following the first puncture (June 8, 1928); the crosses, the curve following the second puncture, fourteen days later; the hollow circles, that following the third puncture, performed after the next seven days.

puncture. Then the tension dropped slowly until it reached the original level from three and one-half to four hours.

The curves for these four patients were almost alike, although there were some differences in the condition of the four eyes. I will consider this curve, therefore, as a standard for comparison.

A great number of cases would be necessary to determine the precise physiologic limits for tension after puncture. One method used was to repeat the puncture on the same eye several times to learn to what extent the tension varied in different states of the eye. Eyes with tabetic optic atrophy (charts 3 and 4) were used and gave the following readings:

CASE 4.—A man, aged 41, had bilateral optic atrophy. Vision in the right eye was 0.03; that in the left was limited to perception of light.

CASE 5.—A man, aged 42, had bilateral optic atrophy, with perception of light only.

In case 4 (chart 3), the anterior chamber was punctured three times at intervals of about three weeks. The three curves were found to be very similar. The first positive readings were obtained thirty minutes after puncture. The original tension was reached in from 88 to 105 minutes. Comparison with the standard curve shows a slightly slower increase throughout. A vascular disturbance of the eye might be assumed. The three curves check well in cases 4 and 5 (charts 3 and 4).

One may conclude that under ordinary conditions, the human eye always answers to the stimulus of the puncture of the anterior chamber in the same way. On the other hand, transient disturbances of the cardiovascular system have a marked effect on the behavior of the intra-ocular tension after puncture.

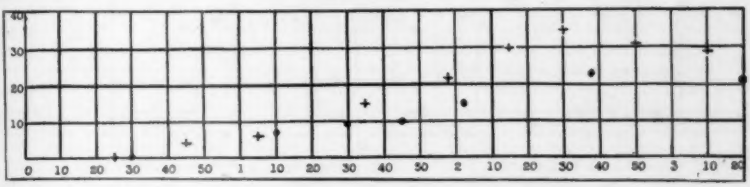

Chart 5.—The tension curve for an eye with arterial congestion. The crosses indicate the curve after the first puncture; the solid circles, the curve after the second puncture when the congestion had disappeared.

T. F., a man, aged 55, showed mesa-ortitis luetica, tabes and optic atrophy of both eyes. One eye had become blind rapidly. The heart was slightly decompensated. When placed in a reclining position, the patient developed an acute arterial congestion of the head; the face became cyanotic, and the blood pressure rose to 180. The puncture was made under these circumstances only because the patient desired it. About two months later, another puncture was performed on the same eye. This time the patient had no circulatory disturbance.

The results are seen in chart 5. The second curve is a great deal like the standard, except that it shows delay in rising. The original level was reached in 122 minutes after the puncture instead of in ninety minutes as in my standard curve. The summit of the curve was found 160 minutes after the puncture. At that time, the tension was 8 mm. higher than before the puncture. Curve 1 shows about the same course: The original tension was reached in 102 minutes, and the summit in 150 minutes, after the puncture. At that time the tension exceeded the original amount by 15 mm. of mercury.

The opposite phenomenon was observed when an eye was punctured under the influence of epinephrine (chart 6). The first positive reading was found sixty-five minutes after the puncture, apparently because

of the vasoconstriction and the tightness of the lining cells due to the epinephrine. The following readings approached more and more the curve for eyes not influenced by epinephrine; yet a difference of a few millimeters of mercury could be observed along the entire curve.

The behavior of the eye may be said to be known under normal conditions, under the influence of arterial congestion and under that of epinephrine.

Further questions arise: What is the significance of these curves? Are they plethysmograms indicating the behavior of the intra-ocular vessels, or are they a measure of the production of fluid after the puncture of the anterior chamber of the eye?

Magitot, in his paper, asks, "Should we still consider the ocular tension as due to the aqueous humor?" He answers this question in the negative. He considers that vasomotor processes are the main causes of the change in intra-ocular tension after the withdrawal of the aqueous. The eyes of animals offer convincing proof of this belief.

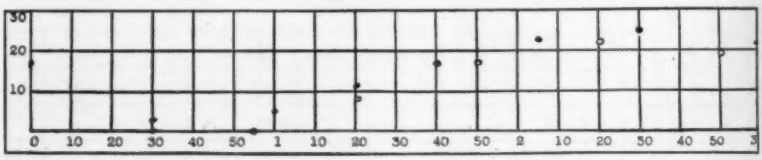

Chart 6.—The tension curve after the puncture of the anterior chamber of an eye under the influence of epinephrine. The solid circles indicate the curve for a normal eye, after puncture; the hollow circles, that for the same eye under the influence of epinephrine.

The two most conclusive observations of Magitot are: 1. The changes of intra-ocular pressure take place in thirty minutes after puncture in the eyes of animals. 2. Transient oscillations of the tension occur after puncture.

But for the same two considerations, I must oppose Magitot's opinion as far as human eyes are concerned. The tension in human eyes rises in an almost straight line, and does not show any oscillations in the first hour. Slight vasomotor reactions may take place during this period, but they are not indicated in the tonometric curve.

After puncture the intra-ocular pressure in the human eye shows a smooth curve. There are no sudden changes in any direction which one would expect if they were due to changes in the caliber of the intra-ocular vessels. The intimate relation between the amount of aqueous in the anterior chamber of the eye and the intra-ocular tension is another reason why I consider the tonometric curve a measure of the flow of fluid into the eye. However, the action of the angle of the anterior

chamber, the safety valve (Duke-Elder [3]), has to be considered. It probably checks the rise of the tension past normal limits. Below this limit there is probably no outflow through the canal of Schlemm. The tonometric curve, therefore, indicates the full amount of reformation of the aqueous.

The amount of inflow depends mainly on the capillary blood pressure and on the permeability of the lining membrane. These two factors are in intimate connection with the caliber of the intra-ocular vessels. They last much longer, and are definitely more pronounced than the primary vasomotor reaction which takes place immediately after the puncture. This point of view explains curves modified by arterial congestion and by epinephrine.

Thus the excess of pressure shown in the curve indicated by crosses over that indicated by dots in chart 5 is due to the increased permeability of the lining cells and to the increased capillary blood pressure. Both factors are more pronounced in the first curve than in the standard curve because the vascular apparatus of the eye was under a severe arterial congestion at the time of the puncture.

In order to explain the curve given by Magitot, more must be known about the state of the patient's eye at the time of the experiment. For example, it is not impossible that in Magitot's case of optic atrophy after fracture of the skull there was an anomaly of the circulation in the sinus cavernosus due to the injury.

REGENERATION OF THE AQUEOUS

The next question which arises is, may any conclusions be drawn about the mode of regeneration of the aqueous from these curves? Hagen observed that the "anterior chamber was restored to full depth four minutes after the puncture while the normal tension was restored after one and a half hours." He concluded that in his cases the fluid which refilled the anterior chamber immediately after the puncture, came from the vitreous body. His first case, however, did not give any information about the regeneration of the aqueous in the normal human eye. From the history and the low tension of this case before the puncture, one may fairly conclude that Hagen was dealing with a case of atrophy of the eye. The theory of Hagen that the primarily regenerated fluid comes from the vitreous body, may hold good in such severely damaged eyes. The patient in case 2 (chart 1) had a choroidal tumor but otherwise had an "almost normal eye." Yet it certainly was not typical that the anterior chamber was restored to normal depth in four minutes. In cases of choroidal tumors, the vitreous body undergoes severe destruction in an early stage. It is easily understood, there-

3. Duke-Elder, W. Stewart: The Nature of the Intraocular Fluids, London, George Pulman and Sons, Ltd., 1927.

fore, that after the emptying of the anterior chamber in this case, free vitreous fluid filled the chamber, especially since the pressure in the vitreous space was slightly increased by the tumor. Case 2 seems authentic as far as the tension after the puncture is concerned, but it cannot be taken as proof that the regenerated fluid is derived from the vitreous body in normal eyes.

Several of Hagen's conclusions seem open to question. First, I have never seen the anterior chamber of a normal eye restored to full depth four minutes after puncture. Second, it is difficult to estimate just when an anterior chamber is of exactly normal depth. One cannot rely on the exactness of one's own judgment. I have therefore measured the volume of the anterior chamber by sucking up the fluid, and I have found conditions to be as shown in the table.

In the three experiments, the aqueous was drawn up in both punctures until the pupil began to contract. The result showed that twenty minutes after the first puncture, the anterior chamber contained only from 50 to 60 per cent of the original volume.

Volume of Aqueous Humor After Two Punctures of the Anterior Chamber

Patient	First Puncture, Volume in Cc.	Interval in Minutes	Second Puncture, Volume in Cc.
1	0.245	20	0.160
2	0.26	22	0.165
3	0.27	25	0.180

Do the 160 cmm. which were found in the anterior chamber of patient 1 twenty minutes after the first puncture correspond to the pressure of the eyeball at that time (from 4 to 5 mm.), or is there any disproportion between the amount of fluid in the anterior chamber and the intra-ocular pressure? Decision is aided by the experiments performed by Koster [4] and by Wessely [5] to determine the amount of fluid which must be injected into the anterior chamber of the eye of a rabbit in order to increase the tension by a given amount. Wessely found an increase of the intra-ocular pressure of 1 mm. of mercury when he injected water in the following amounts: 3 cmm. when the original tension was from 10 to 15 mm. of mercury; 1.5 cmm. when it was from 15 to 20 mm.; 0.8 cmm. when it was from 20 to 25 mm., and 0.7 cmm. when it was from 25 to 30 mm.

So far as I know, there are no corresponding figures for the human eye. I performed a few experiments, injecting 10, 15, 20 and 25 cmm. of physiologic solution of sodium chloride into the anterior chamber of blind eyes with a normal anterior segment and observed the rise of tension. The results checked very well with the figures given by Wessely

4. Koster: Arch. f. Ophth. **41**:30, 1895.
5. Wessely: Arch. f. Augenh. **60**:8, 1908.

for the eyes of rabbits. I was exceedingly interested in the range of values between 0 and 10 mm. of mercury and was amazed to learn that there was no proof that the intra-ocular tension immediately after the puncture was zero.

If the puncture is made with the keratome, the incision is large enough to guarantee free communication and equilibrium between the pressure in the anterior chamber and the atmosphere. In these experiments, however, the holes made with the finest needle obtainable were perfectly water tight and air tight. It might be expected that the intra-ocular tension was close to zero after the puncture. The exact figures, however, were not obtained.

I had an opportunity to measure the tension of a normal eye manometrically immediately after puncture. The patient had a sarcoma of the orbit, and a normal eye with a vision of 1.0 was to be enucleated. The experiment was performed on the day before the exenteration of the orbit. The needle of a Wessely manometer was inserted into the anterior chamber, and the tension was measured for fifteen minutes. The fine puncture needle was then introduced and portions of the aqueous withdrawn slowly at intervals, in order to enable my assistant to readjust the manometer. In three minutes the anterior chamber of the eye was almost empty, and the pupil had begun to contract. The manometer showed an intra-ocular tension of — 5 mm. of mercury. This negative pressure evidently was due to the fact that the cornea and sclera were a little too rigid to compensate for the loss of the aqueous by a corresponding decrease in the volume of the eye.

From the behavior of the eye in the first thirty minutes after the puncture of the anterior chamber, the conclusion may be drawn that during this time the tension rises from — 5 to + 5. How much fluid, then, must have been produced by the ciliary body in order to produce this rise in tension? When an attempt was made in three cases to reinject the aqueous after the puncture without withdrawing the needle, 155 cmm. of aqueous were needed in one case, 170 cmm. in the second and 165 cmm. in the third, in order to bring the tension up to + 5 mm. of mercury. The foregoing amounts of fluid should usually be found, then, in the anterior chamber thirty minutes after the first puncture. The refilling of the anterior chamber can be accomplished, therefore, without any contribution from the vitreous body.

It is a question whether or not the curves shown support the view of Wessely that "the difference in the reaction of human and animal eyes to the puncture is due to the difference in the intensity of the stimulus. The puncture of the anterior chamber means a loss of 14 per cent of the total volume of a rabbit's eye and only 2½ to 4 per cent of a human eye." In other words, Wessely stated that the vascular apparatus in both kinds of eyes reacts in the same way and with the same intensity,

but that the puncture is a stronger stimulus for the eye of a rabbit than for a human eye. The anterior chamber of the eye in a patient with tabetic optic atrophy was punctured twice at an interval of twenty minutes. Altogether, 0.405 cc. of aqueous was obtained, that is, 6 per cent of the total volume of the eyeball. The tension did not rise any faster than after a single puncture. I am unable, therefore, to make any contributions to support this part of Wessely's theory.

SUMMARY

A study was made of the behavior of tension in the normal human eye after puncture of the anterior chamber. Under these circumstances, the following conclusions were made:

1. The intra-ocular tension is a measure of the production of intra-ocular fluid, this production chiefly depending on the condition of the vascular apparatus of the eye.

2. The data found concerning the regeneration of the aqueous confirm, almost throughout, the theory of Wessely that (*a*) there are only quantitative differences between the regeneration of the aqueous in the eyes of human beings and animals, and that (*b*) the second aqueous is always derived from the blood.

3. The process of regeneration of the aqueous is accompanied by marked vasomotor changes in the eyes of animals.

4. These phenomena are less marked in the human eye, probably owing to the greater stability of the vascular apparatus.

EMBOLISM OF SEVERAL BRANCHES OF THE ARTERIA OPHTHALMICA*

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Embolism of the central retinal artery or of one of its branches is a rather frequent occurrence; embolism involving several branches of the arteria ophthalmica, however, seems to be rare enough to justify the reporting of a new case.

REPORT OF CASE

History.—A white man, aged 31, an embroidery worker, eight years previous to examination had had gonorrhea of four months' duration without further complications and without other venereal disease. For several years following this, he had repeated attacks of an eruption of the skin chiefly localized at the outer sides of the legs. He did not have any history of disease of the lungs, and the family history in this respect was negative. He had been married for three years; his wife had never been pregnant; she was sickly. He had not suffered any serious injuries, and did not drink or smoke to any great extent.

On the evening of June 2, 1928, the patient noticed pain and rapidly decreasing vision in the right eye, which to that time had been normal; the next morning (Sunday) the right eye was completely blind. He went to a well known hospital for diseases of the eye and was treated in the dispensary for a few days (with hot applications and atropine). All the routine examinations (roentgenograms of the teeth and of the nose and sinuses, the Wassermann blood test, urinalysis) showed nothing abnormal.

Clinical Picture.—When I saw the patient, on June 9, he presented the following clinical picture:

The right eye was sensitive. There was marked photophobia. Both lids, chiefly the upper one, were edematous. The upper margin of the orbit was somewhat tender to pressure. Some diffuse resistance was felt in the middle of the roof of the orbit, close to the brim.

The conjunctiva was normal, except for slight chronic catarrh. The eyeball showed injection of the ciliary arteries. The movements of the eyeball were free. The palpating glass rod (as used in Dr. Schoenberg's method) revealed a sore spot in the upper temporal quadrant close to the equator of the globe. The cornea was dull, especially in the lower portion. The aqueous was somewhat turbid. The iris was swollen and discolored. A plaquelike swelling was present in the nasal upper quadrant of the small iris circle; here also were some posterior synechiae. The lens was clear. The pupil was irregularly dilated and did not react. Thick, diffuse cloudy opacities appeared in the vitreous; large red spots were made out in the nasal lower periphery of the fundus. Intra-ocular tension was ½. The amaurosis was complete.

The left eye was normal, except for slight myopia. Vision was 20/50; after correction with a lens of —1.5 diopters, it became 20/20.

* Submitted for publication, Dec. 12, 1928.

The sudden onset of the condition indicated either some embolic process or a rapidly progressing axial (retrobulbar) neuritis. Serious objections to the first diagnosis presented themselves. The severe uveitis with its almost papule-like appearance, the hemorrhages in the retinal periphery, the photophobia and the marked pain in the eyeball, together with the circumscribed tenderness of the upper orbital margin as well as of the upper temporal quadrant of the globe, did not seem to fit in with the clinical picture of a simple embolism of the central retinal artery. On the other hand, there was the remote possibility that some inflammatory process in one of the nasal sinuses of the same side had caused an axial neuritis and, at the same time, uveitis and circumscribed orbital periostitis and tenonitis. To be sure, the localization of the sore spot on the eyeball and at the upper orbital margin did not tally with an ethmoiditis or a sinusitis frontalis or maxillaris. However, there hardly seemed to be any other diagnosis open.

Examination.—On June 11, the patient was admitted to the Sydenham Hospital and a thorough general examination was made. I quote the following pathologic observations:

The apex of the heart was in the fifth intercostal space in the midclavicular line. Shocks or thrills were not noted. The organ was not observed to be enlarged on percussion. The sounds were of good quality, without murmurs. The electrocardiogram showed: lead I, P wave 2 and 3 widened; lead II, R wave 3 notching; lead III, T wave 3 inverted; Q. R. S., 1 and 2, high voltage.

The reactions in the Wassermann and Kahn tests were negative. The urine was normal. The gonococcus fixation test was 2 plus (the last made on June 27).

The nose, throat and sinuses were examined by Dr. G. D. Wolf. The mucous membrane on the left side was dry, congested and thickened. The septum had deviated markedly to the left side. The middle turbinate on the right side presented some evidence of ethmoiditis. The tonsillar tissue was enlarged and injected.

The skin as examined by Dr. Rostenberg showed typical erythema nodosum.

Roentgen examination was made by Dr. Unger. The frontal sinuses were large and clear. The ethmoids were clear. A distinct increase of density of the right antrum was observed, strongly suggesting involvement of this region. A dense, round shadow, which appeared to be an outgrowth of tissue in this region, and which appeared to be granulation tissue, was seen. The sphenoids were fairly clear.

The diagnosis was chronic myocarditis, erythema nodosum, involvement of the right antrum, uveitis and retrobulbar neuritis.

Treatment.—The treatment consisted of the use of salicylates, later alternating with atophan; atropine, and hot applications to the eye.

The eye became somewhat less irritated and tender to touch. However, sufficient evidence of a pathologic condition of the nasal sinuses of the right side was present to justify a surgical procedure, and on June 20, at my request, Dr. Wolf opened the right antrum, ethmoid and sphenoid, cleared them out and allowed them to drain. Uneventful recovery followed, except that there was a slight rise in temperature during the following days.

The eyeball showed objectively slow but steady improvement; the injection lessened, the aqueous became clearer, the pupil dilated and the vitreous cleared up, so that on June 22 for the first time details of the fundus could be made out unmistakably; the disk distinctly pale, its outlines blurred, the retinal arteries apparently narrow, hemorrhages plainly to be made out in the lower half of the macular region and in the lower nasal quadrant of the periphery. The intra-ocular tension was slightly below normal.

Spots formerly tender in the middle of the upper orbital margin and in the outer and upper quadrant of the globe were hardly sensitive any more. The complete amaurosis, however, was unchanged.

This total therapeutic failure made us reconsider the whole case. If a septic condition of the sinuses had been to blame for such a rapid and complete loss of vision, treatment by a radical evacuation and drainage of the sinuses should have been at least partially successful.

Second X-Ray Examination.—Another examination was made on June 25 by Dr. E. J. Ryan, this time concentrating on the orbit. It revealed marked infiltration of the right antrum, and in the right orbit a dense, rounded shadow, 0.5 mm. in diameter, on the superior border of the orbit, suggesting the probability of a new growth.

On the same day, a complement-fixation test for gonococcus in the blood proved to be 2 plus.

On June 27, gonococcus vaccine containing 50,000,000 gonoccoci was given hypodermically, without apparent result.

In the meantime, the eyeball had become almost completely pale; the aqueous and the vitreous were clear. The picture of a somewhat atypical embolism of the central retinal artery was more and more evident: pale, not sharp disk; very thin arteries; veins of irregular caliber, and large hemorrhages in the lower half of the fundus.

To complete the examination, a tuberculin test (Pirquet) was made on June 29, the results of which proved negative, and the prostate was explored (by Dr. M. J. Siegelstein). An abstract of his observations follows: The prostate was moderately enlarged, boggy and congested. The vesicles were not palpable. Perivesicular infiltration was not evident. A nodule of an old gonococcal epididymitis was present on the left testicle. The right testicle was normal. The prostate apparently was not the primary focus of the infection responsible for the condition of the eye. The history of past and present erythema nodosum, with a transient systolic murmur heard at the apex and in the tricuspid areas of the heart, and a weakened P wave in lead 2 indicated that the patient had a chronic endocarditis and an associated chronic myocarditis, rheumatic in origin, which was responsible for the embolus of the central retinal artery.

Final Diagnosis.—These observations gave a satisfactory explanation of the whole condition. The primary disease was a "rheumatic" (or possibly gonorrheal) chronic endocarditis and myocarditis. This provided a loosening of its vegetations and multiple emboli or the breaking up of one larger embolus in the arteria ophthalmica with clogging of the arteria centralis retinae, of the arteria supraorbitalis and of at least one arteria ciliaris anterior. This would account for the sudden onset of complete blindness, on one hand, and for the tender spot on the upper orbital margin and the iritis, on the other; very likely, a small vessel supplying the sclera had also been embolized, causing the circumscribed sore area in the upper temporal quadrant of the globe. The presence of retinal hemorrhages, a rather uncommon occurrence in embolism of the central retinal artery, indicated that the emboli were infectious, resulting in a mild septic form of retinitis.

COMMENT

On looking up the literature on similar multiple embolization of other blood vessels complicating the outstanding retinal embolism, I found few cases bearing a marked resemblance to mine.

Leber[1] wrote: "In some cases (of embolism of the central retinal artery) a complication with embolism of vessels of the uveal tract seems to occur; in such instances, the embolus is sometimes of a slightly septic nature so that inflammatory symptoms develop at the same time. Thus combinations of sequels of embolism of the central retinal artery with embolism of the posterior ciliary arteries occur." He cited a case reported by H. Schmidt, in 1874, in which the chief vascular lesion (embolia arteriae centralis retinae) was complicated by an iridochoroiditis. Leber further quoted the reports of cases of Stellwag (1882) and Nuel (1896), respectively, in which iritis and iridocyclitis complicated the original condition and in one of which (Stellwag's case) the other eye later became involved too. The iritis either developed simultaneously with the embolism of the central retinal artery, at times causing severe pain, or followed a short while later. "The iritis may recede; the process thus ends in an optic atrophy, which later may be diagnosed by the ophthalmoscope." (This is another parallel with my case.) "In other instances, a pupillary exudative membrane and thick vitreous opacities may not allow a definite diagnosis. . . ."

Another case which might be cited is one reported by Houwer.[2] He described the postmortem observations in a case of nephritis with thrombosis of the left carotid extending into the cranial cavity. The patient had not been examined clinically or ophthalmologically until just before the terminal stage. An embolism of the central retinal artery was discovered with multiple emboli in the ciliary arteries and in the muscles of the eyeball, and a hemorrhagic infarct in the choroid. The latter observations he said were unique and had not been published before in ophthalmologic literature.

I do not know of other similar cases. The combination of embolism in several branches of the arteria ophthalmica is, however, so rare that it seems worth recording.

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1. Leber: Die Krankheiten der Netzhaut in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, second edition, Leipzig, 1915, p. 196.

2. Houwer, A. W. M.: Arch. f. Augenh. **99**: 240, 1928.

ACUTE HYPOTONY IN RETINAL DETACHMENT*

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LOS ANGELES

Schnabel,[1] in 1876, was the first to describe a peculiar complication in the course of a retinal detachment: the eyeball suddenly became soft to a degree that it could hardly be palpated through the closed lids, the anterior chamber was twice as deep as normal, and the iris seemed to be drawn far backward. Schnabel was under the impression that the thinned wall of a posterior staphyloma (it was a case of high myopia) had burst, and that the contents of the eyeball had evacuated into the orbit. He saw his mistake in a few days, when the eyeball started to regain its normal tension and appearance. Since that time, for a period of more than fifty years, sixteen more cases have been reported by various authors. With the exception of small details in regard to the cornea, which was normal in some cases and which had folds in Descemet's membrane in others, in regard to the pupil, which was either contracted or dilated, and in regard to the ophthalmoscopic observations, the clinical features were strikingly similar in all cases reported. In 1915, Leber[2] therefore felt justified in establishing acute hypotony in retinal detachment as a new clinical entity, notwithstanding the small number of case reports. I had the opportunity to observe two cases of this rather interesting complication.

REPORT OF CASES

CASE 1.—Mrs. M. S., aged 47, who was observed in the Eye Hospital of the University of Kieff in 1923, came under my care complaining of a sudden loss of sight in the left eye which had occurred six days before examination. Ophthalmoscopic examination revealed a considerable detachment of the retina in a highly myopic eye (—16). Complete rest and internal administration of potassium iodide were advised, and a pressure bandage was applied over the left eye. A week later, the patient returned complaining of pain in the left eye. There was a marked pericorneal congestion, the pupil was narrow, the anterior chamber was extraordinarily deep and the iris, which was greatly congested, seemed to be drawn backward, presenting at the same time a distinct tremor. The fundus could not be seen well on account of vitreous opacities, but a dark, solid protrusion from the fundus on the nasal side was suggestive of a choroidal detachment. The ocular tension was so low that it could not be registered by any tonometer, and the movements of the eyeball were accompanied by a distinct folding of the sclera.

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* Read before the Eye, Ear, Nose and Throat Section of the Los Angeles County Medical Association, March 5, 1928.

1. Schnabel: Arch. f. Augenh. **5**:67, 1876.

2. Leber: Die Krankheiten der Netzhaut, Leipzig, W. Engelmann, 1916, p. 1422.

In a few days, under ordinary treatment (with atropine, salicylates, etc.), the condition started to clear up: the uveal inflammation subsided, and the tension returned to normal.

CASE 2.—M. Z., a man, aged 35, who was observed in 1928 and presented to the oculists of the Eye and Ear Hospital of Los Angeles, always had poor vision in the right eye, which was "nearsighted." A year before examination he had entirely lost the sight in this eye, and five days before that time he started to feel pain in the right eye. The examination revealed a mild pericorneal congestion, an extremely deep anterior chamber and a trembling iris. At the periphery of the iris a bulging, due to the ciliary processes, could be noted, particularly when the eye was examined with the slit-lamp. The fundus could not be seen on account of vitreous opacities. The eyeball was unusually soft, and on palpation gave a peculiar "spongy" impression. No tonometric measurement could be taken.

The dominating symptom in these cases, as well as in the rest of the group, is extreme hypotony. There is only one pathologic condition that I can think of which presents a similar decrease in ocular tension, and that is a recent and large perforation of the sclera with an extensive loss of the vitreous; in this condition one can hardly feel the presence of an eyeball through the closed eyelids.

Next comes the condition of the anterior chamber; it is much deeper than the one seen after cataract extraction; the iris has a distinctly concave surface, and on its periphery one can see the impressions formed by the ciliary processes. A similar extreme deepening of the anterior chamber is found only in the late stage of a plastic cyclitis when the iris is drawn far backward by the organized exudate, but contrary to this condition in acute hypotony, the iris is freely movable and as a rule presents tremor.

A distinct uveal involvement with opacities in the media, a congested iris and pericorneal hyperemia have been described in every case of acute hypotony. The pain accompanying the uveitis varies with the degree and character of the inflammation, but it hardly ever justifies the enucleation of the eyeball. It should be remembered that acute hypotony is a transient condition, and one will find expectant and conservative treatment the most satisfactory. It was the lack of experience in early days which prompted some authors to remove the eyeball after the onset of hypotony.

If knowledge concerning this peculiar condition, understanding of its clinical symptoms, its course and outcome are of practical value, the determination of the origin of acute hypotony is a matter of scientific importance.

It is interesting to note that every symptom of acute hypotony which has just been analyzed is only an extreme exaggeration of symptoms characteristic of retinal detachment proper. A mild degree of hypotony is a condition most frequently found in retinal detachment. Some

authors (Schnabel, Walter[3]) stressed the frequency of this symptom even before the tonometer was used. Recently, Kümmel,[4] Baurmann,[5] Gonin,[6] Lagrange[7] and numerous other authors proved the presence of this symptom with the tonometer. A slightly deepened anterior chamber in retinal detachment was first demonstrated by Schweigger[8] and Samelsohn;[9] it was also mentioned by Fuchs;[10] and does one not see mild forms of iritis with an occasional posterior synechia or cyclitis and some tenderness in the ciliary region in the majority of cases of detached retinas?

The striking similarity of the clinical symptoms in "acute hypotony" and in retinal detachment proper goes further than that. In the case of Ginsberg and Simon,[11] as well as in case 1, the detachment of the choroid was diagnosed ophthalmoscopically; it has also been found in all cases examined pathologically. There is reason to believe that when this detachment has not been noted by the clinician, it has been obscured by vitreous opacities. On the other hand, it is a fact not fully appreciated, although established long ago by Müller[12] and emphasized later by Meller,[13] that in retinal detachment the suprachoroidal space contains the same fluid as the subretinal space, or, in other words, that detachment of the retina is always accompanied by detachment of the choroid. In the light of this peculiar analogy, it seems to be possible that "acute hypotony" as a whole is only an extreme exaggeration of a retinal detachment, and an inquiry into its origin is justified as an attempt to clarify the pathogenesis of retinal detachment; it is possible that, when acting under "normal" conditions, the same forces that produce an acute hypotension are responsible in a less degree for a detachment of the retina. There is also the advantage of studying facts in an exaggerated state, when their correlation can be established easier.

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3. Walter: Klinische Studien über Netzhautablösung, 1884, Inaug. Diss., Zürich.

4. Kümmel: Arch. f. Augenh. **95**:214, 1925.

5. Baurmann: Klin. Monatsbl. f. Augenh. **80**:214, 1928.

6. Gonin: Société française d'ophthalmologie, May, 1920.

7. Lagrange, F.: Du glaucome et de l'hypotonie, Paris, Gaston Doin, 1922, p. 377.

8. Schweigger: Bericht über die 14 Versammlung der Ophthalmologischen Gesellschaft Heidelberg, Munich, J. F. Bergmann, 1882, p. 34.

9. Samelsohn: Bericht über die 14 Versammlung der Ophthalmologischen Gesellschaft Heidelberg, Munich, J. F. Bergmann, 1882, p. 40.

10. Fuchs, E.: Lehrbuch der Augenheilkunde, Vienna, F. Deuticke, 1926.

11. Ginsberg and Simon: Zentralbl. f. prakt. Augenh. **22**:161, 1898.

12. Müller, L.: München. med. Wchnschr., 1903.

13. Meller, J.: Arch. f. Ophth., vol. 80, p. 170.

One indisputable fact in regard to the origin of "acute hypotony" is the sudden and great decrease in the volume of the vitreous body. This is the only explanation of the simultaneous retraction of the diaphragm (the iris and lens) and of the fall of intra-ocular tension. However, I cannot agree with Hirshberg [14] that the vitreous in these cases undergoes an actual shrinking or coagulation. Not only is this assumption against the present understanding of the vitreous structure and especially of its changes in a high degree of myopia and in senility, but it would not be possible for a shrunken vitreous to regain its normal size and consistence in a few days.

The hypofunction of the ciliary body due to cyclitis was held responsible for acute hypotony by Lauber [15] and Vogt.[16] As Leber pointed out, however, any decrease in the production of intra-ocular fluid is regulated immediately by a decrease in the outflow, and no considerable loss of tension can follow. Besides, it must be remembered that acute hypotony never occurs in any condition but retinal detachment, and any sensible explanation of this phenomenon must take into consideration factors existing only in retinal detachment. In the majority of cases of retinal detachment, there is direct communication between the liquefied vitreous and the choriocapillaris. A hole in a detached retina has been demonstrated by numerous authors (De Wecker,[17] Leber,[18] Nordenson,[19] and others). It is logical to admit that under certain conditions large amounts of the liquids of the vitreous may be absorbed by the large surface of the choriocapillaris. Pressure through a pressure bandage or even through the action of a spastic orbicular muscle, a change of the osmotic conditions of either side of the choroidal system and perhaps other as yet unknown factors will precipitate a sudden passage of liquids from the vitreous into the choroidal blood vessels and result in acute hypotony. Recently Prof. J. Meller [20] proved pathologically that in a case of intra-ocular hemorrhage the ciliary epithelium participated in the absorption of blood, and he concluded that the function of the epithelial cells of the ciliary body and probably also of the retina and ora serrata may be "the absorption of fluid from the vitreous and its transmission along the lymph cells." If this is true, the epithelial lining of the vitreous chamber may be another channel for a sudden absorption of fluid with a resulting hypotony.

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14. Hirshberg: Zentralbl. f. prakt. Augenh. **22**:207, 1898.
15. Lauber: Ztschr. f. Augenh. **20**:118, 1908.
16. Vogt: Klin. Monatsbl. f. Augenh. **72**:335, 1924.
17. De Wecker: Traité complet et d'ophthalmologie, 1889, vol. 4, p. 145.
18. Leber (footnote 2, p. 1349).
19. Nordenson: Die Netzhautablösung, Wiesbaden, 1887.
20. Meller, J.: Significance of Ciliary Epithelium in the Absorption of Vitreous Hemorrhages, Arch. Ophth. **57**:134 (March) 1928.

CONCLUSIONS

1. Acute hypotony, characterized by a great decrease of ocular tension, a sudden deepening of the anterior chamber and a mild form of a uveitis, is a definite clinical entity.

2. It occurs only in retinal detachment.

3. The prognosis in acute hypotony is favorable, and the treatment should be conservative.

4. Acute hypotony is probably the result of sudden absorption of fluid from the liquefied vitreous.

THE ETIOLOGY OF SYMPATHETIC OPHTHALMIA

FURTHER NOTES *

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OMAHA
AND
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CHEYENNE, WYO.

In 1924, von Szily [1] reported experiments with herpes virus from herpetic keratitis, with which he produced uveitis in the eyes of rabbits, which was transmitted to the opposite eye, giving a clinical and histologic picture resembling sympathetic ophthalmia. In 1926, we [2] reported experiments which confirmed his work, and gave added evidence that the process in these animals was one of extension in the nerves and chiasm to the opposite eye. Von Szily had advised inoculation of uveal tissue from eyes enucleated for sympathetic ophthalmia into a subscleral pouch in animals. We suggested that the chances were against the production of an involvement of the second eye by this method, as it succeeded with herpes virus in only 10 per cent of cases, and the occurrence of the involvement evidently depended on a number of circumstances. In the absence of ordinary bacteria, the production of uveitis in the eye into which tissue had been inoculated would strongly suggest the presence of some virus. The production of keratitis on corneal inoculations would also be suggestive. It must be remembered that in the case of herpes the inoculable virus disappears rapidly from the original lesion, so that, as Goodpasture has shown, it is difficult to obtain positive inoculations from the cornea of a rabbit from five to six days after a positive inoculation. Hence, negative results would not be surprising from eyes that were removed weeks and months after the disease had begun, as is usually the case in sympathetic ophthalmia.

In order that the theory of a filtrable virus as the cause of sympathetic ophthalmia and the importance of trying inoculations from every eye removed for sympathetic ophthalmia may not be forgotten, we report the results of inoculations made from four enucleated eyes from undoubted cases of sympathetic ophthalmia. Most of the results were negative; in one case, however, a lesion was produced, which is at least interesting.

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* Submitted for publication, Dec. 7, 1928.

* From the Department of Ophthalmology, University of Nebraska Medical College.

1. Von Szily: Klin. Monastabl. f. Augenh. **72**:593, 1924.

2. Gifford, S. R., and Lucic, L. H.: Tr. Sect. Ophth. A. M. A., 1926, p. 20.

REPORT OF CASES

CASE 1.—The first case was reported in our previous work. It was that of a man, aged 70, whose left eye had been iridectomized elsewhere five weeks before. A rapidly progressive uveitis developed in the right eye, and when the left eye was enucleated, it showed a large melanosarcoma of the choroid surrounded by infiltration characteristic of sympathetic ophthalmia. As the eye had been operated on, the case cannot be classed as one of spontaneous sympathetic ophthalmia which is known to occur rarely in eyes with melanosarcoma. Uveal tissue was inoculated into the ciliary pouches of ten rabbits and into the scarified corneas of three others. Negative results were observed in all but one, which showed a keratitis for three days. This was not progressive, however, and was considered as possibly due to the scarification alone.

CASE 2.—A boy, aged 15, seen with Dr. Harold Gifford and Dr. Patton, developed sympathetic ophthalmia eleven months after a nail injury, with prolapse of the iris; the uninjured eye showed a hazy cornea and numerous corneal precipitates. The injured eye was at once enucleated, following which the other eye improved; however, a secondary glaucoma developed, which required two punctures of the iris. The tension was finally controlled, and at the time of dismissal the vision was 20/30. Sections of the enucleated eye showed infiltration of the choroid and iris, which was characteristic of sympathetic ophthalmia.

Uveal tissue from this eye was used for inoculation into six rabbits; in two animals, it was inoculated into the pouches between the ciliary body and the sclera; in two, into corneal scarifications; in one, into the chiasm, and in one, into the vitreous. The two rabbits in which tissue was inoculated into the ciliary pouches showed a moderate reaction, which subsided after two weeks without producing visible plastic exudate, and which was probably attributable to the operation alone. One corneal inoculation produced a fairly marked keratitis, with infiltration at the site of inoculation and congestion of the iris. This subsided, however, in five days and no inoculations were made from it. The other inoculations showed negative results.

CASE 3.—Keith L., a boy, aged 3, was seen by the courtesy of Dr. McConkie of Carroll, Ia., one month after a knife injury to the right eye, with the picture of well developed sympathetic ophthalmia in the other eye. Enucleation was done at once, and the material was used for the inoculation into both corneas of two rabbits, and for intratesticular injection into another.[3] All showed negative results.

CASE 4.—Mrs. H. was referred to in a paper on iridencleisis as the patient in whom the incision was made somewhat too deep, and the iris sphincter was not cut, which resulted in a large subconjunctival prolapse of the iris and the ciliary body, part of which was later excised to reduce a recurrence of tension. One month later, a few cells could be seen in the other eye by the slit lamp, and slight blurriness of vision was noted. The cells increased gradually, blurriness of the disk developed, and the left eye was removed, although vision was still 20/100 and tension normal. The right eye cleared up rapidly and has remained quiet, with vision 20/20. Sections showed infiltration of the ciliary body with large mononuclear and epithelioid cells.

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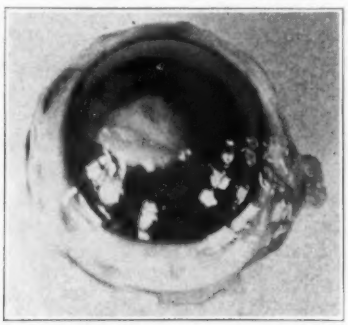
3. This inoculation was made with the idea of possibly increasing the virulence of any virus present so that secondary inoculations would be more definite, as was shown to be possible by Rivers and Gillett with the virus of chickenpox, J. Exper. Med. **38**:673, 1923.

Fresh uveal tissue was inoculated into the ciliary pouches of the eyes of two rabbits with negative results; the tissue was also inoculated into three corneas, of which two showed negative results. The third corneal inoculation, however, in rabbit 3, resulted in progressive deep keratitis. The superficial wounds healed in seven days, but the congestion continued and the central opacity increased in size and density, covering the upper half of the cornea. Congestion was still marked when the eye was enucleated after four weeks, as shown in the illustration. Two weeks after inoculation, the cornea was scarified in one small area to obtain material for inoculation into the cornea of two other rabbits. In one cornea, this produced a keratitis which lasted one week, but was not progressive and left only a slight scar; in the others, the reaction was even less marked. Uveal tissue which was preserved three weeks in glycerin did not produce a reaction on corneal inoculation. The progressive keratitis produced in rabbit 3 with this material, considered as a result, is open to one objection, which must be stated. Whereas the results of examinations for bacteria in uveal material and corneas were negative in the other cases, in this animal after two weeks, scrapings of the cornea showed intracellular diplococci which were not present in the uveal tissue. Thus the keratitis might be attributed to the presence of this organism in the rabbit's



Eye of rabbit inoculated in case 4.

conjunctival sac. It was also found in the cornea of rabbit 5, which showed a transient keratitis. In view of the extreme difficulty of producing keratitis with most organisms by corneal scarification, and the rapidity with which control scarifications heal when exposed to invasion by conjunctival organisms, it would seem more likely that this organism was a secondary invader, and not the cause of the keratitis. The keratitis produced in the second animal, which subsided in a week, may have been due to this organism.

Other attempts at inoculation of material from persons with sympathetic ophthalmia, following von Szily's suggestion, may be briefly summarized. As stated in our previous work, Marchesani [4] inoculated tissue from one undoubted case into ten animals; in three the inoculations were intradural and in seven they were made into the ciliary pouches; the results were negative in both series. Volmer [5] and Meesman and Volmer [6]

4. Marchesani: Klin. Monatstabl. f. Augenh. **75**:218, 1925.
5. Volmer, discussion of Marchesani (footnote 4).
6. Meesman and Volmer: Am. J. Anat. **92**:271, 1927.

used material from five cases for inoculation into the ciliary pouches of sixty rabbits. Material from one of the cases produced uveitis in the second eye of three rabbits. Two of these were found to have tuberculosis elsewhere, but the third, which developed a diffuse uveitis with exudate in the anterior chamber, did not show any other lesions post mortem. They considered the histologic picture characteristic of herpetic uveitis rather than of sympathetic ophthalmia, and believed that it was possibly a stall infection from other animals with herpes.

Undelt [7] inoculated into the cornea of two rabbits, and into the ciliary pouch of another, material from a fresh case of sympathetic ophthalmia, with negative results.

In discussing our paper, Knapp [8] mentioned one case of sympathetic ophthalmia from which material was inoculated into a ciliary pouch with negative results.

Marchesani [9] carried out experiments with herpes virus on twenty-seven rabbits, similar to those of von Szily, but did not produce uveitis of the second eye. He reported [10] a case of infection of one eye with *Bacillus subtilis*, in which the second eye developed uveitis. The organism was found in the first eye in sections and cultures, and the histologic picture was that of chronic uveitis resembling the picture of sympathetic ophthalmia. Inoculations into animals produced a chronic uveitis with a similar histologic picture, but the second eye was never involved.

The experimental work of von Szily has been confirmed by Abe [11] and Velhagen, Jr.[12] The former, by various methods of inoculations with herpes genitalis, produced uveitis of the second eye in 30 per cent of his animals. Sections demonstrated the route of transmission by the chiasm and optic nerves. This was also evident from the invariable occurrence of papillitis in the second eye before any other signs of inflammation, and from the fact that a series of animals which had previously had one optic nerve resected did not show any involvement of the second eye after a later inoculation. Abe mentioned several cases of sympathetic ophthalmia in which distinct meningo-encephalitic symptoms preceded the involvement of the second eye, and quoted Takahashi's observations of inflammatory changes in the spinal fluid in four cases of sympathetic ophthalmia. He pointed out some differences from sympathetic ophthalmia in the histologic picture seen in his animals. Velhagen produced involvement of the second eye only in animals in which the first eye showed papillitis; this is further evidence of the direct nerve transmission of herpes.

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7. Undelt: Klin. Monatsbl. f. Augenh. **75**:825, 1926.
8. Knapp, discussion of Gifford and Lucic (footnote 2).
9. Marchesani: Am. J. Anat. **97**:575, 1926.
10. Marchesani: Am. J. Anat. **97**:557, 1926.
11. Abe, T.: Arch. f. Ophth. **117**:375, 1926.
12. Velhagen, K., Jr.: Klin. Monatsbl. Augenh. **78**:894, 1927.

In 1926, von Szily [13] reviewed the pros and cons of the virus theory, describing Guillery's work with tuberculous and other toxins, which he was unable to confirm. He described one of the second eyes involved, in which the condition was allowed to run its course for two years; at that time the end-result was much like that seen in some cases of sympathetic ophthalmia with cataract, detached retina and bone formation in the choroid. He emphasized again the fact that his work is only a method of imitating the clinical and histologic picture of sympathetic ophthalmia; the claim is not made that the herpes virus is the cause of sympathetic ophthalmia, but that the production of this picture by such a virus is highly suggestive of the production of sympathetic ophthalmia by a virus, as yet undiscovered, via the sheaths and chiasm of the optic nerves. He stated that negative results of direct inoculation from sympathetic ophthalmia are to be expected, as it is not known how long the etiologic agent of sympathetic ophthalmia remains viable, and one is not able to take material for inoculation from the active foci, as is possible from corneal lesions.

It is unneccessary to repeat the reasons why we consider the theory of a virus as the cause of sympathetic ophthalmia the most plausible of the theories put forth. However, if the cause is a virus, the greatest difficulties in the proof of this by direct inoculation into animals would seem to lie in two factors: the short length of time during which the virus is inoculable and the small amount of virus probably present in extremely small foci. The most hopeful avenue of approach to this problem would seem to be a search for methods of enriching the virus in artificial mediums, such as Noguchi's medium or glycerol, as has been done with the herpes virus, or in tissue cultures, or by passage through suitable living animal tissues, as was done with the virus of chickenpox. Inoculations could then be made by different methods with a larger amount of active virus.

TESTS WITH UVEAL PIGMENT IN SYMPATHETIC OPHTHALMIA

In 1921 and 1922, Woods [14] reported investigations on the immune bodies in the serums of patients with injuries of the uveal tract. In cases of injury that ran a favorable course immune bodies developed, as shown by a positive reaction to complement-fixation tests. Patients with severe chronic uveitis resulting from such injuries gave negative reactions, as did three who showed signs of sympathetic ophthalmia; the others showed photophobia and one, transient congestion. Of two patients with old cases of sympathetic inflammation, only one developed true sympathetic ophthalmia; the others showed photophobia and

13. Von Szily: Deutsch. med. Wchnschr. **52**:1598, 1926.

14. Woods, A. C.: Tr. Sect. Ophth. A. M. A., 1921, p. 105; Arch. Ophth. **51**: 451, 1922.

one, transient congestion. Two patients with old cases of sympathetic ophthalmia gave negative reactions. Patients with various types of nontraumatic uveitis gave reactions. Woods believed that if a positive test has not developed within six weeks after the injury, this is one indication for enucleation. Woods and Knapp [15] reported the case of one patient with sympathetic ophthalmia, with a negative reaction, who was treated by desensitizing doses of uveal pigment; this treatment was beneficial, and the complement-fixation test became positive. The patient had given a positive intradermal reaction before receiving treatment. Recently, Woods [16] mentioned that he had treated ten patients with sympathetic ophthalmia in this way. Two of them were later operated on for cataract, with a slight reaction.

Whether or not one is an adherent of the anaphylactic theory, the report of such a test, which would help to decide whether or not an eye should be enucleated in order to forestall sympathetic ophthalmia, seemed well worth investigation. Previous workers, quoted by Woods, had obtained conflicting results; Kummell found uveal antibodies in the serum of patients with sympathetic ophthalmia.

Recently, Fodor [17] reported the results of complement-fixation tests in six cases of sympathetic ophthalmia and in a large number of cases of other diseases of the eye, including traumatic and nontraumatic iridocyclitis. He found that the patients with sympathetic ophthalmia gave a negative reaction to complement-fixation tests. In other cases, however, the reaction varied; cases of penetrating wounds which ran a favorable course showed a negative reaction more often than a positive one. In retinitis pigmentosa, in cases of which Woods had obtained positive reactions, Fodor obtained two positive and two negative reactions. He concluded that the reaction gives no definite indication of the probable course of uveitis, and that a decision as to whether or not an eye should be enucleated on the basis of this test would be dangerous, as it might allow the retention of a sympathogenic eye.

Dr. Woods sent us some of his pigment antigen, and we tested two cases of undoubted sympathetic ophthalmia and three cases of traumatic iridocyclitis which ran a favorable course. As in Dr. Woods' cases, the cases of sympathetic ophthalmia showed negative reactions to complement-fixation tests, but they also showed negative reactions to intradermal tests; they gave no evidence, therefore, of the sensitization which he had observed in sympathetic ophthalmia. The other three cases showed negative reactions to skin tests. Complement-fixation tests were not performed in the last cases, as we were especially interested in the clinical application of the skin test.

15. Woods, A. C., and Knapp, A.: Arch. Ophth. **51**:560, 1922.
16. Woods, A. C.: Arch. Ophth. **57**:488, 1928.
17. Fodor, G. I.: Pigment Reactions in Perforating Wounds, Klin. Monatsbl. f. Augenh. **79**:759, 1928.

While conclusions cannot be drawn from these few cases, they show that hypersensitiveness, as indicated by the skin test, is not the rule in sympathetic ophthalmia. This need have no great bearing on the anaphylactic theory, as the processes of immunity and anaphylaxis are complicated ones, and by no means completely understood. These tests should be tried in a much larger number of cases, so that their value may be adjudged.

CONCLUSIONS

1. Material from four cases of sympathetic ophthalmia was used for inoculations into animals.

2. Corneal inoculations from cases 1 and 2 produced mild keratitis in two rabbits, but this was too slight to be considered as showing the presence of a virus. From case 4, a severe progressive keratitis was produced, indicating the presence of an organism or virus. Organisms found after two weeks may have been secondary invaders.

3. Inoculations into ciliary pouches were considered negative, although the operative reaction was severe enough to mask slight reactions which may have occurred.

4. Methods of enriching the possible etiologic agent before inoculation are suggested.

5. Cases of sympathetic ophthalmia were found to give negative reactions to complement-fixation tests, as well as to skin tests.

THE ENTOPTIC PHENOMENON OF THE BLUE ARCS

A STUDY OF THE SECONDARY EXCITATION IN THE RETINA*

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The visual phenomenon usually referred to as "the reddish-blue arcs and the reddish-blue glow of the retina" is one that has attracted the attention of several investigators, who are not in agreement as regards its explanation. Therefore it seemed worth while to undertake further experimental investigations of this unique phenomenon.

The arcs may be observed under the following conditions: When, in a dark room, a small rectangle of light is made to fall on the temporal side of the macula, near the fovea and near or in the region of the median line, there are seen, circling the fixation point, two glowing blue arcs which have their origin at the upper and lower edges of the rectangle of light, and which terminate converging toward the blind spot. Recently there has been considerable discussion concerning the cause of this phenomenon. The very nature of the phenomenon presents unusual obstacles to an experimental investigation, and the few demonstrable facts have led to two entirely unrelated theories accounting for its appearance. Further progress toward an explanation awaits the presentation of more facts that will confirm one theory or the other or that indicate the errors in both hypotheses. The study herewith presented is an experimental review of previous determinations of facts and conditions relating to the phenomenon, together with further experimental observations and deductions.

Amberson [1] gave an excellent summary of the literature on the subject previous to 1924. Since the writing of his paper, work has been done by Davis,[2] Ellis [3] (1927), Ladd-Franklin [4] and others. Amberson

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* From the Physiological Laboratory, Princeton University, Princeton, N. J.

1. Amberson, W. R.: Secondary Excitation in the Retina, Am. J. Physiol. **69**:354, 1924.

2. Davis, Hallowell: The Theory of "Visible Radiation from an Excited Nerve Fiber," Science **67**:69, 1928.

3. Ellis, F. W.: Studies in the Physiology and Psychology of Visual Sensations and Perceptions, Am. J. Physiol. **5**:462, 1901; Secondary Excitation of the Retina and the Variation of the Intensity of the Resulting Sensation, ibid. **82**:290, 1927.

4. Ladd-Franklin, Christine: The Reddish Blue Arcs and the Reddish Blue Glow of the Retina. See Your Own Nerve Currents Through Bioluminescence, Proc. Nat. Acad. Sc. **12**:413, 1926; Visible Radiation from Excited Nerve Fiber, Science **66**:239, 1927.

explained the phenomenon on the basis of a secondary excitation, and was supported in this view by Davis and Ellis. Ladd-Franklin believed that the evidence indicated an explanation on the basis of a visible radiation from an excited nerve fiber. In support of this theory, she offered evidence which will be discussed later.

APPARATUS

The apparatus used in my investigation consisted of a large box set up on a table and lined with black paper to prevent any reflection and to cover any defects in the sides of the box. Curtains of heavy black cloth were hung over the open side of the box, so that it was not necessary to work in a completely dark room. A hole was cut in the back of the box, and so constructed that various filters and diaphragms could be attached and interchanged at will. A lamp box with a lens producing parallel rays was placed on a stand outside the box so that the light was directed on the diaphragm placed over the hole. In this fashion any light that leaked out from the lamp box was prevented from reaching the observer's eye.

It was found necessary to have a fixation point that would not give any light and yet would itself be visible; such points were made by drawing out glass tubing to a fine capillary, then dipping the tip of the capillary into collodion and phosphorescent zinc sulphide. This technic gave a point 1 mm. in diameter on the end of the glass rod, which could then be mounted in any desired position, with the advantage of permitting instant change of position when such change was needed. On illumination for a few seconds with an ordinary flashlight, these points glow brightly and remain visible for two or three minutes, and yet do not give enough light to interfere with the other features of the experiments. This type of fixation point is more suitable than the one used by Amberson, which consisted of a fixed point of luminous paint, because it can be moved about much more freely. This feature is essential for certain experiments to be described.

OBSERVATIONS

Areas of Retina Involved.—My first investigations related to the question of the area of the retina involved in the primary stimulation.[5] This area was found to be an elliptical section of the retina, situated entirely on the temporal side of the fovea, beginning at its temporal edge, the long diameter of the ellipse extending outward 15 degrees. A second area, which on stimulation gives the "blue spike" effect, consists of a narrow ring from 2 to 5 degrees concentric with the fovea. Since these two areas overlap, it is possible, by moving the fixation point so that the stimulating beam of light passes from one area into the other, to produce a transition from one effect to the other, showing that both are manifestations of the same phenomenon. When the stimulating beam is divided,

5. "Primary" refers to the system consisting of the external conditions and that part of the nervous system and the sense organ involved in perceiving, or reacting to, these external conditions. "Secondary" refers to all the remaining conditions connected with the phenomenon, which are due to the existence of, or action of, the primary system.

so that the larger part is directed to the "arc area" and the smaller part to the "spike area," both effects may be produced simultaneously. It is impossible to obtain the arcs on stimulation of any part of the fovea. Amberson suggested that this central area (fovea), which on stimulation does not produce the arcs, corresponds with the rod free area of the retina, and hence that arc and spike effects are rod rather than cone phenomena. This conclusion is justifiable; it must be borne in mind, however, that it applies to the primary system only; the secondary system is not correspondingly limited to the rod system, but may equally well involve the cone system.

The area [6] of the retina involved in the secondary effect, the blue arcs and the spike, is limited to the region between the blind spot and the temporal edge of the region of primary stimulation. The distribution

Variation in Size and Character of the Arcs with Change in Region of Primary Stimulation

Distance of Fixation Point from Slit, Mm.	Distance Between Arcs at Maximum, Mm.	Distance of Normal to Curve at Maximum, from Fixation Point, Mm.	Character of Arc
5	9	20-25	Spike, bright edges, filled with blue haze
10	17	22-25	Distinct arc, slight haze within
15	25	23-25	Same
20	33	25	Arc bright, haze along inner edge
25	42	25	Arc bright, sharp edges
30	51	26	Arc less bright, thinner
35	60	26	Arcs quite thin, not bright
40	68	26	Arcs are fine lines, faint
45	..	..	Arcs merely suggested, very indefinite
50	..	..	No arcs

* The distance from the point of fixation to the approximate center of the blind spot (projected) was 130 mm.

of the nerve fibers in this region, determined in our experiments by measurements of the arc pattern, using a fixation point in various positions as indicated in the accompanying table, does not leave any doubt that the pattern of the arcs is identical with the pattern of the nerve fibers. Wagener and Gipner [7] and Ronne [8] gave charts of the nerve pattern; a comparison of these charts with those made from the arc patterns proves that they are identical. Amberson offered further proof in an elaborate table of comparison of the absolute length of the arcs with papillofoveal distance.

The results given in the table permit one to carry a step further Amberson's conclusion that the rods are the retinal nerve endings

6. For maps of these areas, see Amberson: Am. J. Physiol. **69**:354, 1924.

7. Wagener, H. P., and Gipner, J. F.: Visual Fields in Localized Chorioretinitis of Jensen Type, Arch. Ophth. **55**:479 (Sept.) 1926.

8. Ronne, Henning: The Different Types of Defects of the Field of Vision, J. A. M. A. **89**:1860 (Nov. 26) 1927.

primarily involved. Since the fovea is composed entirely of cones, while each successive region toward the periphery contains fewer and fewer cones, and since it has been shown that the arcs become narrower and fainter the farther away from the fovea they originate, it may be concluded that the arcs themselves are in some way connected with the cones (as suggested by Amberson on the basis of the color of the arcs), even though the primary system of stimulation appears to be based on rod phenomena alone. These observations and deductions may be summarized by the statement that the primary system is dependent on the function of sensory nervous pathways arising at rod end organs; the secondary system is dependent on the function of sensory nervous pathways arising at cone end organs. It must be borne in mind, however, that the evidence on which these deductions are based is indirect, and not conclusive.

Constancy of the Blue Color.—In order to make sure that the color of the arcs was constant, the blue coloration of the arcs and spike was examined by the following method.

A double diaphragm was set up in such fashion that by using separate filters one slit could be made a different color from the other. Two fixation points were used. By placing a screen between the eyes, one eye was made to observe one slit while the other eye observed the other slit. The natural attempt of the eyes to converge and focus simultaneously on the same object caused consciousness of first one and then the other. This action permitted a quick and repeated contrast of the arcs produced by two differently colored stimuli, and thus a comparison could easily be made between them. Many filters, passing the important colors of the spectrum, were used in the diaphragms, and the resulting arcs compared with each other in this manner.

In no case could the arcs from a slit of one color be considered different in color from those produced by a differently colored slit. A red color in the stimulus appeared at first glance to be an exception to this rule, because the arcs produced in this case were by far the most brilliant, and contained the most color.[9]

The purplish or reddish-blue color which the arcs assumed when the stimulating slit was very bright red appeared to be an additive property of the two colors. When the light of the stimulus was cut down, the purple color changed to the normal blue because the red component was decreased while the blue component remained constant.

To provide a check on these observations, an additional series of experiments on the color of the arcs was made, in which, instead of the lamp and filter system previously described, a quartz mercury vapor

9. In demonstrating the arcs, red is the color usually used for the stimulating beam of light because it affords the best background on which to project the blue arcs.

lamp was used with filters, especially designed for the lamp, these filters transmitting monochromatic light of the various desired wave lengths. The filters used were Wratten filters no. 22, yellow, no. 50, violet, no. 62, green and no. 77, green. It was found that with the yellow filter in the lamp, given a primary stimulus of that color, arcs rich in the normal blue color were obtained. Purple was not observed. The green filters also produced arcs without any purple in them, though the blue was not so rich (saturated) as in those from the yellow. The violet did not produce arcs. A comparison of the arcs from the yellow filtered mercury lamp and those from the red filtered ordinary lamp showed that the arcs were noticeably more purple in the latter case. These observations seem to confirm those from the previously described experiments; to wit, that the color of the arcs is blue, the saturation being variable. Hence it is a mistake to call the arcs "the reddish-blue arcs" or "the purple arcs," since they are in reality blue, and only appear reddish blue or purple under certain conditions.

The Variation in Intensity.—That the arcs vary in intensity with variation in the intensity of the primary stimulus was proved by Ellis (1927),[3] who concluded that because of this variation in intensity, the sensations do not follow an all-or-none law, as Troland [10] believed. This evidence appears conflicting, but a repetition of the experiments showed the arcs varying in intensity below a certain maximum, which was considerably above the minimum intensity necessary to evoke the arcs. (Ellis stated that he could observe three distinct intensities within this range.) Above this maximum, the intensity of the arcs was constant over a considerable range. Experiments were made in which Ellis' system of successive contrast was used but instead of the resistance in the lamp circuit which could be suddenly cut out, fractional transmitting filters were used. Also a graded filter wedge was used, which afforded the advantage of a smooth transfer from one value to another without any lag in the system. This possible lag might introduce a considerable error, for reasons that will be pointed out later.

Ellis' argument that the all-or-none relationship does not hold for the arcs appears based on evidence that has nothing to do with the question. It is a well known fact that a case of nerve action current has not yet been found in which the amount of action current (potential difference) in the conducting nerve varied with variation in the stimulating conditions. On the other hand, instances are on record in which it is shown that with variation in the intensity of the stimulus of an end organ, the frequency of the discharge in the afferent nerve varies

10. Troland, L. T.: The "All or None" Law in Visual Response, J. Optic. Soc., America **4**:100, 1920.

(Adrian and Zotterman, 1926 [11]). There is no direct evidence that this is true in afferent nerves from visual receptors.[12] It seems logical, however, to assume that in this sense organ, as in all others thus far studied, a variation in the intensity of the primary stimulus causes a variation in the frequency of the discharge in the afferent nerves; the variation in the secondary system is, accordingly, the result of changes of frequency. Hence the variation in intensity does not constitute a demonstration that the all-or-none law is violated in this phenomenon, and so is not an argument against considering this phenomenon one due to nerve action.

In my experiments, the variation in intensity of the arcs was first studied with the use of white light in the stimulus system. When the white light was bright, arcs could not be obtained, but as the light was gradually cut down by moving the graded filter wedge across the stimulating slit, the arcs appeared, faint and small at first; they became brightest when the light was nearly all cut out. The same effect was produced with any monochromatic light in the stimulating slit: the arcs appeared brightest just above the threshold intensity of light necessary to produce them. But with monochromatic light an increase in intensity far above the threshold did not cause the arcs to disappear; throughout such an increase, their intensity remained fairly constant. The disappearance of the arcs when white light was used appeared to be due either to fluorescence in the eye or to the fact that the background became too full of light, masking the effect by a primary stimulation over the secondary stimulation.

Under proper conditions, an after-image of the arcs could be seen; this after-image consisted merely of dark bands against the less dark background. It could be produced under only the most ideal conditions: from one to two minutes of adaptation to dark, a wide stimulating slit, a very bright light and a saturated red color in the slit. On the other hand, the after-image of the slit itself could be seen as often as desired.

The state of adaptation to dark of the observer's eye at the moment of stimulation influenced the intensity of the arcs. They appeared brightest to an eye that had been adapted to dark for about one minute;

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11. Adrian, E. D.: The Impulses Produced by Sensory Nerve Endings: I, J. Physiol. **61**:49, 1926. Adrian, E. D., and Zotterman, Yngve: The Impulses Produced by Sensory Nerve Endings: II. The Response of a Single End Organ, ibid. **61**:151, 1926; The Impulses Produced by Sensory Nerve Endings: III. Impulses Set Up by Touch and Pressure, ibid. **61**:465, 1926. Adrian, E. D., and Matthews, Rachel, The Action of Light on the Eye. Discharge in the Optic Nerve, ibid. **63**:378, 1927.

12. Direct evidence recently appeared in the experiments of Adrian on discharges in the optic nerve of the eel and the frog (Adrian and Matthews: J. Physiol. **63**:392, 1927). He stated, "The number of points is not great enough to allow the equation of the curve to be stated, but the frequency is evidently some exponential function of the intensity. Such a relation is not surprising. . . ."

further adaptation to dark caused a slight decrease in the intensity of the arcs. The difference was not large, although the intensity of the arcs was noticeably below its maximum to an eye that had been adapted to dark for only three or four seconds. Therefore, in order to secure uniformity in the records in the experiments next to be described, two minutes was allowed for the adaptation to dark preceding the taking of each record.

There was one fact in connection with the phenomenon of the blue arcs which appears to deserve more attention than has been paid to it by previous investigators. The arcs were elusive, disappearing almost at once if any attempt was made to scrutinize them. When a steady and constant gaze was maintained directly on the fixation point, they disappeared in less than a second. The only way it was possible to keep them in existence was by a constant winking or shifting of the eye or the light. Amberson stated, as quoted by Ellis, in connection with this fact, that "the effect (of the blue arcs) is transitory, fading out in about one second, to reappear when fixation is slightly shifted or the eye is winked."

The Duration of the Arcs.—I undertook a study of the duration of the arcs, using an electrical method of recording this time under varying conditions of stimulation.

A kymograph was set up, to which were applied two signal magnets and an electrical tuning fork with a period of one-fiftieth second. One lever, the lower one in the records, was connected in the lamp[13] circuit in parallel. Since alternating current was used in the lamp at 110 volts, an aluminum rectifier and resistances were connected in series with the signal magnet, so that direct current at about 6 volts was produced for the signal magnet. Only one phase of the alternating current could be utilized by this method, giving a vibratory signal instead of a smooth line in the record, but such a record is sufficient for indicating when the current in the lamp is on. The top lever (signal magnet) was connected with a telegraph key and two dry cells; the key was placed in the observation box convenient to the observer's hand. In making the records, an assistant operated the kymograph and the switch of the lamp circuit, while the observer closed the telegraph key when the arcs appeared and opened it when they disappeared.

An analysis of the records made with this instrument shows that with complete fixation and all parts of the stimulating system fixed, the arcs had a duration of about eight tenths of a second. Figure 1 presents examples of records from which these data are taken. Adaptation to dark for two minutes before the taking of each record brought about an approximate uniformity. In figure 2 a series is shown in the taking of which there was an adaptation to dark for two minutes and only one second between the disappearance of the arcs and the next

13. A neon-filled lamp was used as the source of light, as such a lamp comes to full brilliance immediately on closure of the circuit.

stimulus. In this case, the duration was progressively shorter, the first being six tenths, the second fifty-five hundredths and the third five tenths of a second. Figure 3 shows this effect more plainly, only five tenths of a second elapsing between the disappearance of the arcs and the next

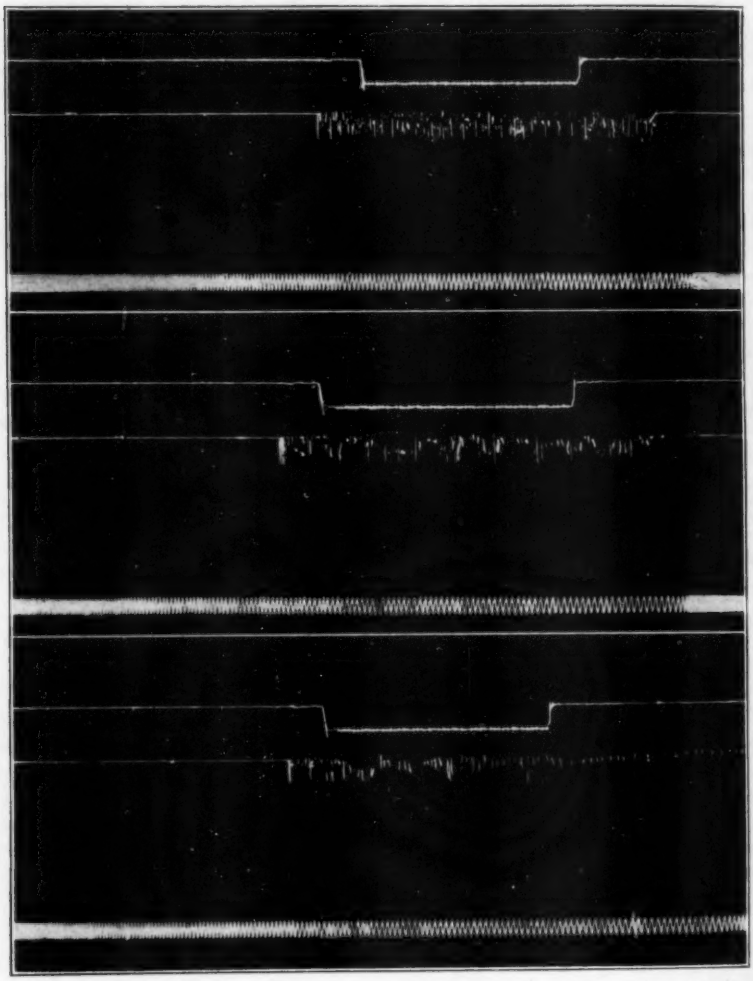

Fig. 1.—Three records taken of the duration of the blue arcs with complete fixation. These records were taken under exactly the same conditions. A two minute period of adaptation to dark preceded the taking of each record.

stimulus. The first had a duration of nine tenths, the second of five tenths and the third of only two tenths of a second.

The remaining records (figs. 4 to 7) show the effect of some motion in the system. In all cases, the duration of the arcs was greatly

increased, in most cases beyond the time interval that could be recorded on the drum (about eight seconds). The series of graphs showing the effect of vibration of the fixation point are interesting. When the fixation point was vibrating faster than the eye could follow, so that the

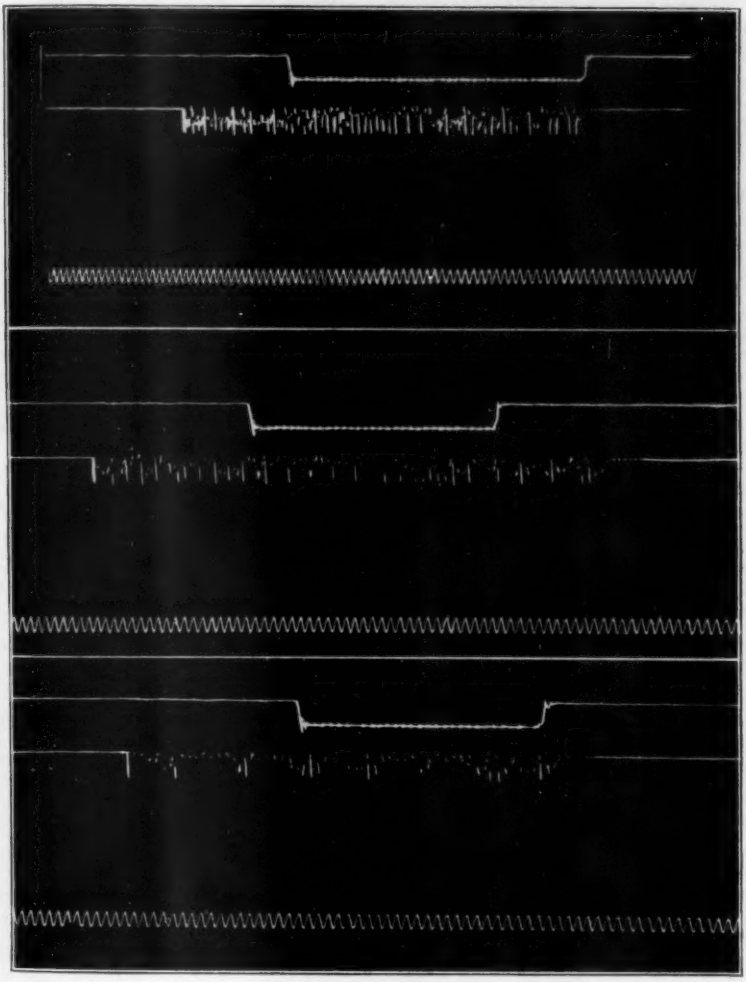

Fig. 2.—Three records of the duration of the blue arcs with complete fixation, with one second's interval between the stimulations.

eye did not move, the duration of the arcs was about normal, eight tenths of a second (fig. 4, *A*). When the vibration of the fixation point was slowed down to the point at which the eye began to move in following it, the duration of the arcs was increased to one and two tenths seconds (fig. 4, *B*). Still slower vibration increased the duration

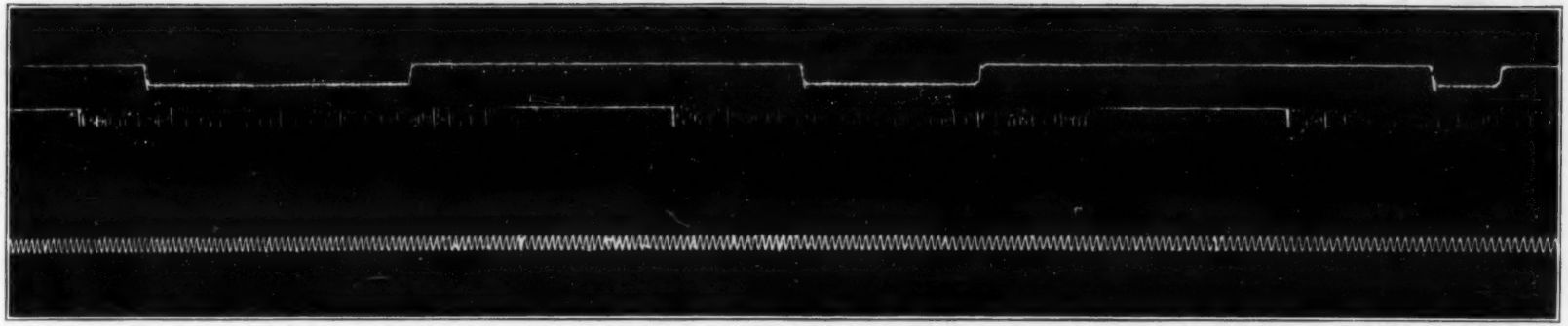

Fig. 3.—A record of the duration of the blue arcs, with complete fixation; fatigue. Time, one-fiftieth second. Adaptation to dark followed by a rest of one-half second was allowed between the first and second and between the second and third stimulations.

enormously, as shown in figure 5, *A* and *B*. In terms of the mechanism of the eye underlying these facts, a complete fixation meant that the primary stimulus was falling constantly on the same sense organs; hence, the number and the identity of the nerves carrying the stimuli so set up did not change, and consequently only one set of nerves or sense organs was stimulated. On the other hand, any movement caused the light from the stimulating slit to fall on first one set of sense organs and then another, thus setting up stimuli first in one nerve group and then in another. Thus, at different moments different nerves were

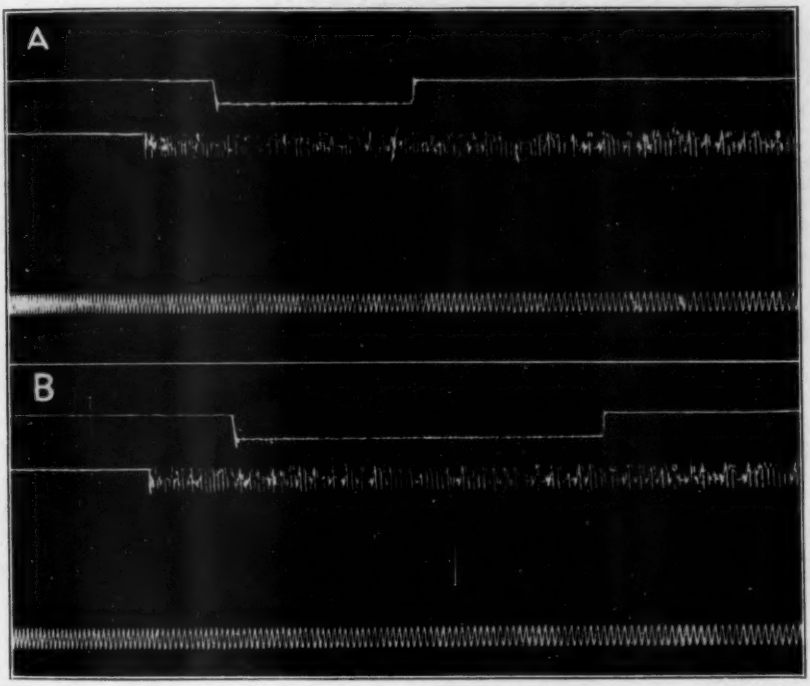

Fig. 4.—*A*. Duration of the arcs with the fixation point vibrating rapidly through a range of 2 mm. *B*. Duration of the arcs with the fixation point vibrating somewhat more slowly than in the previous record, through a range of 2 mm.; time, one-fiftieth second.

involved in the secondary system, and each nerve or end organ secondarily involved received intermittent stimuli instead of a steady flow.

COMMENT

It seems advisable to begin the consideration of these experimentally determined facts by studying the two new pieces of evidence: the results of the experiments on color, and the results of the experiments on duration of the arcs. An explanation of the latter based on the normal

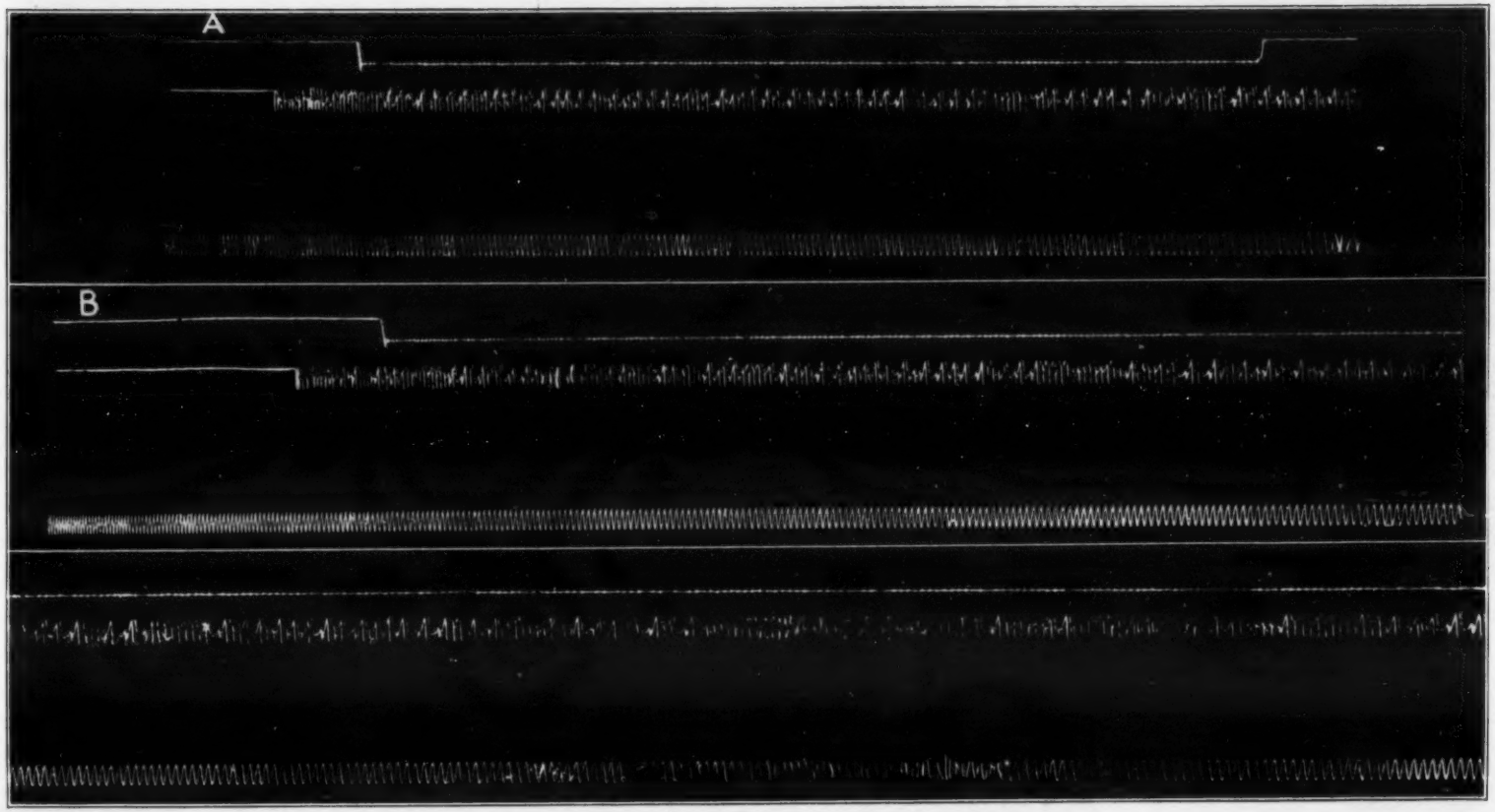

Fig. 5.—*A*. Duration of the arcs with the fixation point vibrating rapidly, but still more slowly than for figure 4, *B*. There was a two-minute period of adaptation to dark preceding each run; time, one-fiftieth second. *B*. Duration of the arcs with the fixation point moving slowly up and down and the eye stationary. These are like the runs shown in figures 4 *A* and *B* and 5 *A*, except that the vibration of the fixation point was slowed down to one vibration per second; time, one-fiftieth second.

and the eye stationary. These are the ... to one vibration per second; time, one-fiftieth second.

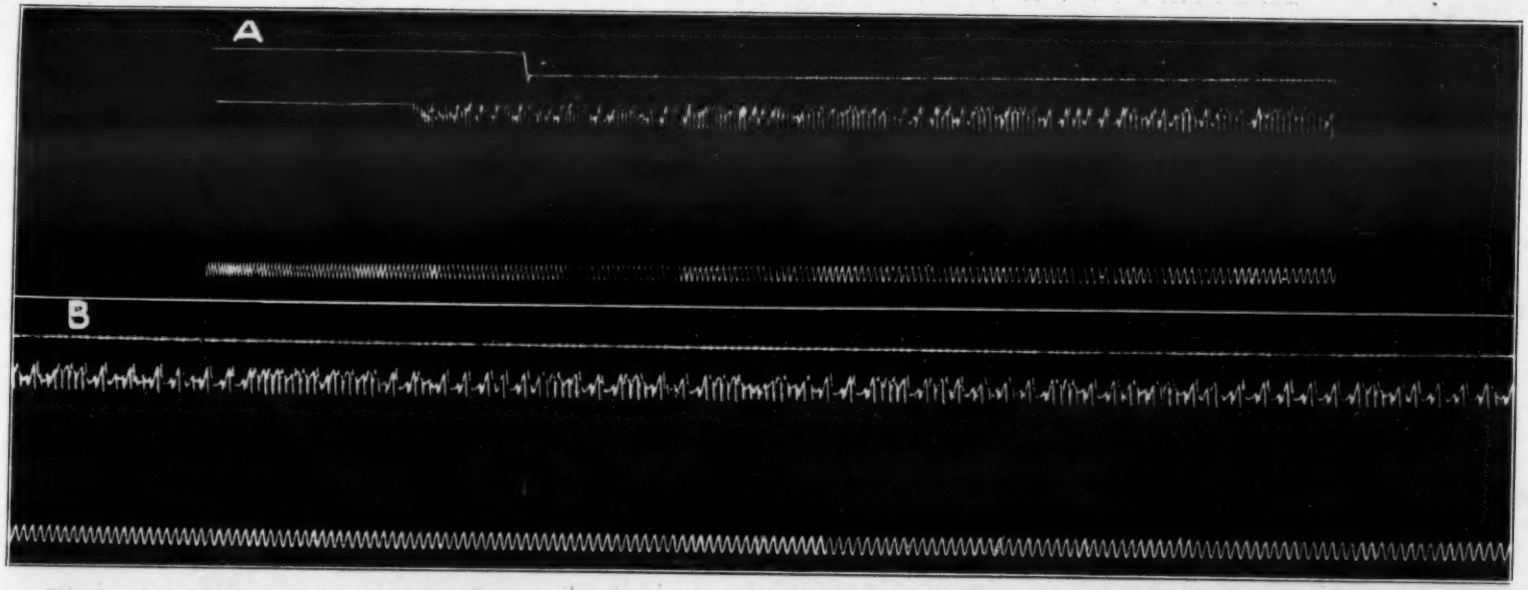

Fig. 6.—*A*. Duration of the arcs, with the stimulating slit moving slowly to and from the fixation point. *B*. Continuation of the record in *A*. The distance from the fixation point to the stimulating slit varied from 2 to 3 cm., moving approximately once a second, or 1 cm. per second; time, one-fiftieth second.

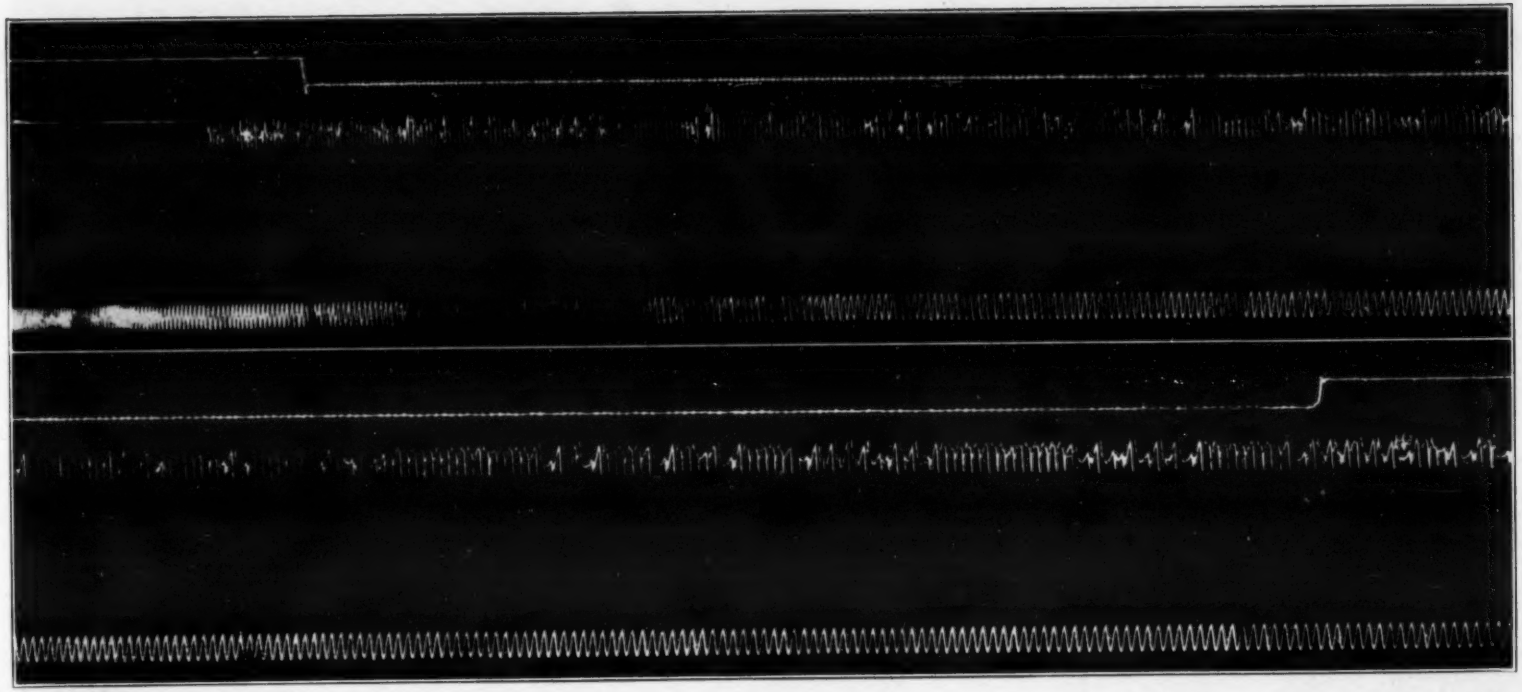

Fig. 7.—Duration of the arcs with complete fixation, except for a slight rotary motion of the head. The illustration shows the first half and the second half of the same record. Two minutes of adaptation to dark preceded the making of the run; time, one-fiftieth second.

behavior of sensory end organs in general appears to have the strongest arguments in its favor. From the work of Adrian and Zotterman (1926) [11] the behavior of a single end organ under a constant stimulus is known. He studied the receptors in the sternocutaneous muscle of the frog and in the pad of the toe of the cat by means of the action currents in the afferent nerves from these end organs. He found that, in the case of the muscle of the frog, "the impulses set up by a single end organ occur with a regular rhythm at a frequency which increases with the load on the muscle and decreases with the length of time for which the load has been applied. The adaptation which occurs when the stimulation is continued is due, in part, at least, to a fall of excitability and it is unlikely that there is much change in the refractory period." As a result of his experiments on the cat, he stated, "When pressure is applied to the disc (a glass disc placed in contact with the footpad of the cat), a discharge of impulses takes place which is at a maximum as the pressure is increasing and falls off rapidly when the pressure is maintained at a constant value. The frequency of the impulses varied with the intensity of the stimulus." In both cases, he found that the size of the individual action currents did not vary, hence that an all-or-none relationship held between the stimulus and the impulse. These two cases, showing a similar action of end organs of entirely different types, and requiring for explanation only the properties of excitable tissue in general, suggest an explanation [14] for the similar behavior of a third type of sensory end organ, the eye. Adrian suggested, in the conclusion of his second paper on the impulses produced by sensory nerve endings: [11] "It is probable, therefore, that other types of end organ will be found to react in much the same way to the particular stimuli which call them into play." Thus, if one is justified, as appears more than probable, in assuming that visual end organs have a decline in frequency of discharge when stimulation becomes constant, the fading in intensity of the arcs and their disappearance within one second are explained. Intensity was shown varying directly as the frequency; hence, as the frequency of discharge of end organs into the nerves of the primary system dropped, the frequency and consequently the intensity of the secondary discharge dropped accordingly. The fact that the secondary discharge ceased completely was due to the near-threshold value of the stimuli from the primary system on the secondary system. The all-or-none relationship was not violated.

14. This explanation has now experimental facts to support it, namely, the work of Adrian: J. Physiol. **61**:49, 1926. In one paragraph, Adrian stated, "In some preparations with small areas and faint lights the discharge fails altogether after one or two seconds."

Before going any further in this comment, I wish to point out that the stimulation of the secondary system by the nerves of the primary system appeared to be of the nature of a secondary excitation, rather than a visible radiation as suggested by Ladd-Franklin. The action currents,[15] pulsating in nature, of the nerves from the sense organs primarily stimulated acted as stimuli to adjoining nerves, setting up in these secondary nerves stimuli that rose to consciousness as the blue arcs. In terms of this mechanism, the constancy of the color of the arcs was the result of a similarity of nature of the nerve impulses; whatever color the primary stimulus might be, the nature of the action current aroused in the secondary system was the same. The intensity of the arcs was dependent on the frequency of the nerve current in the secondary system, which in turn was dependent on the frequency of the discharge in the primary nerves, the latter frequency being dependent on the intensity of the stimulating slit and the state of adaptation of the primarily stimulated end organs. Since the arcs were near the threshold to begin with, a decline in the frequency due to adaptation or to lowering the intensity of the primary stimulus did not have to be large to cause the intensity of the arcs to fall below the intensity or the frequency necessary to bring them to consciousness. There is one question in this connection for which there is as yet no satisfactory answer: Why should the falling frequency of the primary stimulus of almost any color remain above the threshold, while exactly the same frequency in a secondary nerve giving the sensation of a constant blue color falls below the threshold? The answer probably rests somewhere in the mass of ignorance regarding color vision.

In considering the question of the color, I can only suggest the direction from which an explanation might come. The color is not and cannot be a property of the primary system. No matter how the conditions of the primary system were varied, the color of the arcs remained constant. Moreover, the type of sensation that is received by the brain is known to be dependent on the pathway by which it comes; hence it can be concluded that one is dealing with a restricted set of nerve arcs, more like each other in the stimuli to which they are normally responsive than like other sets of visual nerve arcs. Since the sensation aroused when they are stimulated is one of blue light, it is possible that they are afferent nerves from visual end organs sensitive to light of the wave length that one perceives normally as blue. This paragraph is offered merely as a suggestion; it would be eminently worth while if an experiment could be devised to determine accurately the basis of the color of the arcs.

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15. Chaffee; Bovie, and Hampson: The Electric Response of the Retina, Am. J. Phys. Optics **6**:224, 1925.

Troland [10] thought that the decline of the secondary effects is due to the approach to constancy of the electrical response in the primary system, thus making the electrical effect ineffective as a stimulus according to the DuBois Raymond law. It is now known that the pulsating nature of the normal discharge from an end organ is sufficient to produce stimulation in accordance with this law. Amberson called the transitory nature of the arcs a phenomenon of fatigue, or an "adaptation in the direction of decreased sensitivity of ganglion cells" secondarily stimulated. If the argument presented in the preceding paragraph is correct, then the fading of the arcs was an indication of the adaptation or decline in frequency of the discharge of the end organs primarily stimulated. In order to determine whether the adaptation might not be taking place in end organs stimulated by a visible radiation from a nerve, the following experiment was performed. A piece of white paper was placed over the region of the box on which the arcs were projected, and the outline of the arcs traced on this paper. This outline was then transferred to a sheet of black paper, and painted on with a mixture of collodion and phosphorescent calcium sulphide. The black paper was then mounted in the box so that the design of the arcs fell in exactly the same place as the projection of the arcs produced by the stimulating slit. The calcium sulphide was then illuminated for a brief time, so that the intensity of the calcium sulphide illumination appeared about equal to that of the projected arcs. When the stimulating slit was lighted, the projected arcs fell just outside the arcs painted on the background, this condition being determined by the position of the fixation point. The stimulating slit was turned on and the point fixated in the usual manner. It was noted that both the real and the projected arcs disappeared at the same time. Several observers were asked to make the observation, and all stated that in their opinion both arcs disappeared at the same time. If the projected arcs were due to a visible radiation from the excited primary nerves, one might have expected them to fade in much less time than the real calcium sulphide arcs, because while the calcium arcs constituted a steady stimulus, in the projected arc system the adaptation taking place in the primarily stimulated end organs would cause a decrease in the stimulation of the secondarily involved end organs which would multiply the effect of the latter's normal adaptation. There was yet one further characteristic of the arcs which this experiment brought out which gave an indication of the nature of the arcs. With the fixation point so arranged that the projected arcs fell exactly on top of the painted ones, the eye was fixated on the point till the painted arcs disappeared; the stimulating slit was turned on, and the blue arcs jumped out in exactly the same place in which the white ones had appeared a moment before, and where (speaking in terms of the projected field) they were still sending light waves

at an intensity exactly similar (as was a condition set by the experiment) to that of the blue projected arcs. In the previous experiment it was shown that the two types of arc appeared one on top of the other when the proper end organ was adapted for neither; hence, it might be expected that if both arcs were due to light waves striking visual end organs, the new stimuli from the arcs produced by the stimulating slit would be added to those already present, for which the end organ was at the moment adapted, and that both would appear. The fact that only the projected blue arcs appeared, while the painted white ones remained out, corroborates the conclusion reached in the foregoing paragraphs that the arcs could not have been due to a visible radiation, but were caused by a secondary excitation in accordance with the mechanism previously described.

In conclusion, it is essential to answer the objections that have been raised against the explanation of the existence of the arcs on the basis of a secondary excitation. In the first place, it has been stated that the existence of an after-image of the arcs indicated that visual end organs were involved. It has been shown here that the after-image of the arcs occurred rarely, and was then so faint that color could not be perceived in it. Such behavior would hardly be expected if the visual organs were always involved. It seems rather to have been an indication that the visual end organs (of the secondary system) were rarely, and then only slightly, disturbed by the secondary stimulation that produced the phenomenon of the blue arcs, The other objection, that a secondary excitation would have given the wrong "place-coefficient," is invalid if one accepts the evidence of Amberson, who stated that his observers found the arcs in exactly the same place as the "place-coefficient" demanded that they should be. I repeated Amberson's experiments and obtained similar results; it is unnecessary to describe these experiments further, since they are fully set forth in Amberson's report.

Both of the theories led to the expectation that in experiments on subjects with myelinated nerve fibers, the effect would not appear. In the case of visible radiation, the myelin sheath would act as a screen, keeping the radiation from being effective as such. In the case of secondary excitation, the myelin sheath would act as an insulator, as well as a protection for the nerve, keeping the action currents confined to the one nerve pathway.

There are two points that the theory of visible radiation as an explanation of the blue arcs does not cover: (1) the report of Amberson that an observer color blind to blue in one region of the eye could still see the arcs in their normal blue color in that same region of the retina; and (2) the fact that experiments dealing with adaptation of the end organs that might be involved in the phenomenon indicate that end

organs other than those on which the light from the primary stimulating slit falls, are not involved. Unless these objections can be shown to be imaginary, the explanation of the occurrence of the arcs as due to a secondary excitation alone appears to cover the facts in a satisfactory manner. For absolute certainty one must await further knowledge of retinal and visual physiology.

SUMMARY

1. The entoptic phenomenon of the blue arcs and the blue spike is described, and the method used in producing it for study outlined.

2. The area of the retina involved in the different parts of systems making up the phenomenon is discussed, and a table given showing the results of measurements of these areas.

3. A study of the color of the arcs is presented, with evidence to show that the arcs are actually blue and not purple as has been stated, and that the color is constant.

4. The variation in intensity of the arcs is discussed, and evidence is offered indicating that the all-or-none law does hold, and is not violated as Ellis indicated.

5. The duration of the arcs is presented, measured by a graphic method; it is shown that the fading of the arcs can be explained on the basis of the normal adaptation of sensory end organs in general.

6. It is concluded that the theory of secondary excitation offers a satisfactory explanation of the facts, while the theory of visible radiation does not hold in the light of the new evidence presented.

RING ABSCESS OF THE CORNEA*

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AND
C. E. HUNT, M.D.
OMAHA

The fact that we have had the opportunity to see four typical cases of ring abscess at the Methodist Hospital during the years 1927 and 1928 seems to justify a brief report of these cases.

REPORT OF CASES

CASE 1.—Rudolph W., aged 27, was referred to Dr. H. Gifford on Nov. 20, 1927. The day before, while working in a well, he was struck in the left eye by a piece of steel from the pipe wrench which he was pounding. The lids were so swollen that the cornea could be seen only when the lids were separated with lid hooks. The conjunctiva was chemotic, and the eye almost fixed. The cornea showed a complete ring of yellowish infiltration, 1.2 mm. wide, a clear zone of 1 mm. separating this from the limbus. The surface of the cornea was slightly rough, but no wound could be seen. Evidently, the steel, which was shown by x-ray examination to be in the vitreous, had entered through the sclera, the wound being concealed by the chemotic conjunctiva. No light perception was present. Through a scleral incision just behind the limbus, a piece of steel, 3 by 1.5 mm., was extracted with the inner pole magnet. Ten cubic centimeters of milk was given intramuscularly, although the temperature was already 101 F. The next morning, the signs of panophthalmitis were even more marked, the cornea inside the ring of infiltration being completely opaque. Evisceration of the globe was performed, the cornea being excised as close to the limbus as possible. The vitreous was replaced by turbid, slightly blood-stained fluid. No frank pus was seen in the anterior chamber.

Smears from the cut surface of the cornea and from the vitreous showed large, gram-positive bacilli, which were especially numerous in the vitreous. Blood plates obtained from both the cornea and the vitreous showed a pure culture of a similar bacillus. This organism was gram-positive, motile, formed spores and grew in rapidly spreading, confluent, grayish colonies which were typical of *Bacillus subtilis*.

CASE 2.—Mr. H., a patient of Dr. H. B. Lemere, was struck in the left eye by a piece of steel from a hammer. Twenty-four hours later, examination showed a well developed panophthalmitis. Near six o'clock, there was a perpendicular wound in the limbus, about 4 mm. in length. The iris was torn at its lower attachment; a band of infiltration, 2 mm. wide and 2 mm. from the limbus, extended completely around the cornea. The remainder of the cornea was only slightly hazy. Despite vigorous treatment, the condition became rapidly worse, and the eye was eviscerated.

Examination of smears and cultures taken from the crushed cornea and vitreous body showed the predominating organism to be *B. subtilis*.

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CASE 3.—George S., the day before admission, was struck in the eye by a nail which he was pounding. When first seen, the eye gave evidence of a beginning panophthalmitis. There was a ring of gray infiltration completely surrounding the cornea, 1.5 mm. from the periphery. There was a ragged wound in the cornea which extended through the limbus. The vitreous was cloudy. The next day signs of panophthalmitis were more marked and the eye was eviscerated. A section of the cornea was taken for histologic examination.

Bacteriologic examination of smears and cultures from the cornea and vitreous showed a gram-positive organism of varying lengths, which was motile, and in which no spores could be found. It grew aerobically in large greenish-gray colonies. With it was a small gram-positive coccus which could not be separated.

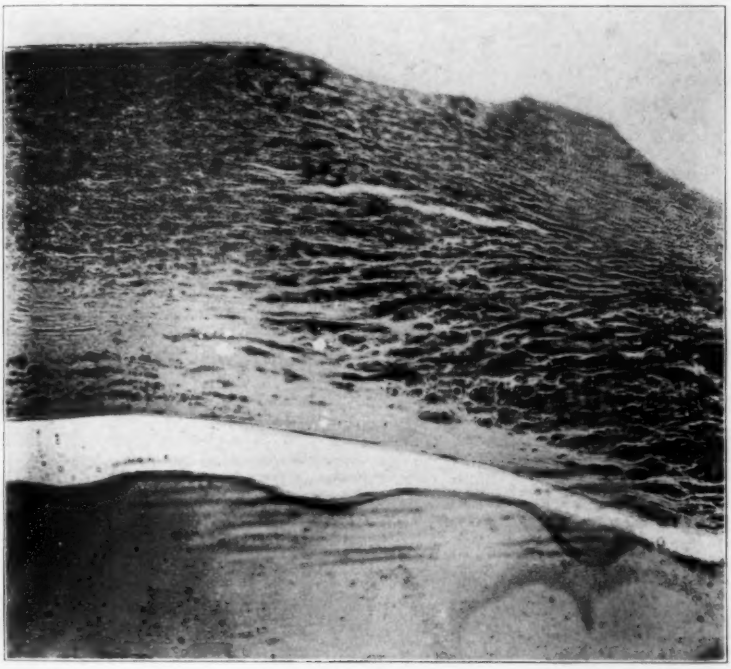

Fig. 1.—Section of cornea in case 1.

This organism could not be identified definitely but was probably a member of the subtilis group.

CASE 4.—Mr. S. was injured by a stick of wood. Twenty-four hours later examination showed a small penetrating wound at the limbus. The anterior chamber was full of blood. There was no infiltration of the cornea. One day later, there was a definite ring in the deeper layers of the cornea near the limbus. The next day the whole cornea was opaque, and panophthalmitis was present. The eye was eviscerated. Bacteriologic work was not done.

COMMENT

Histologic examination of sections from case 1 showed the infiltration to be densest in the middle layers of the cornea. The epithelium

and endothelium were absent, but Bowman's and Descemet's membranes were left intact. In the center of the cornea the infiltration was less marked, but at the periphery where the cornea had swollen to three times its normal thickness, the infiltration was greatest and extended almost through the cornea. The cells were almost exclusively polymorphonuclear.

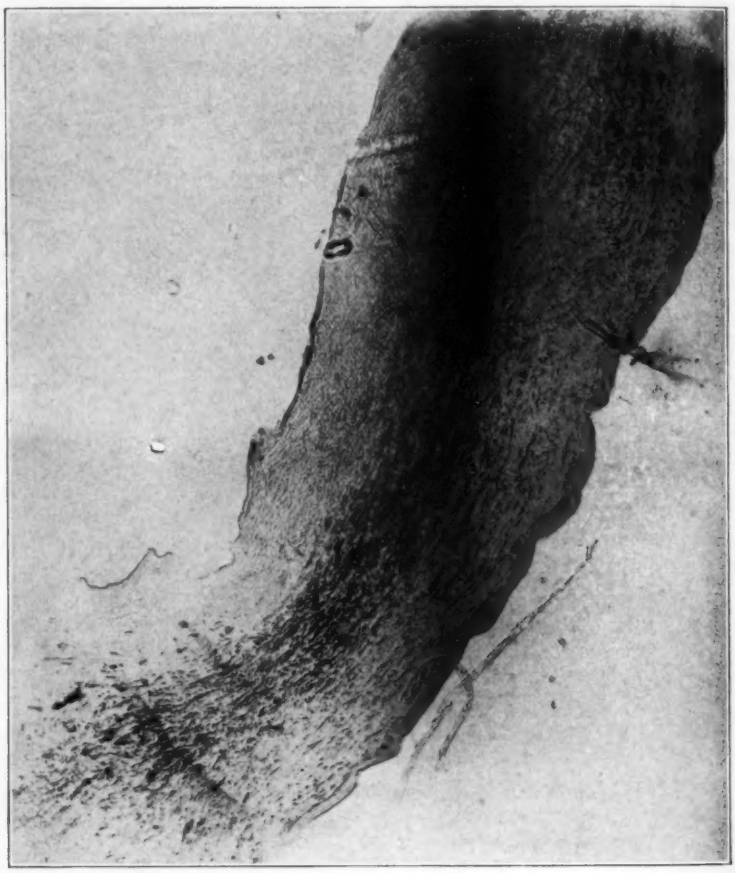

Fig. 2.—Cornea of rabbit inoculated with culture from case 1.

Sections stained with Verhoeff's modified Gram stain showed gram-positive bodies of varying shape, chiefly intracellular, which were probably bacilli undergoing degeneration.

The corneal wound was included in the section of case 3. The infiltration extended from this wound into the deeper layers of the cornea. Bowman's and Descemet's membranes were intact, as in the previous cases, except at the point of injury. The anterior chamber was filled with

pus and fibrin. The remainder of the section showed an extensive panophthalmitis.

Three rabbits were inoculated with a suspension of *B. subtilis* from case 1. In one, the bacteria were introduced into the anterior chamber, in another into the vitreous of an eye, the iris of which had been traumatized with iris forceps through a corneal incision. In the first case, a transitory reaction resulted; in the last two, panopthalmitis developed on the second day. The cornea of the eye in which the iris had been torn developed a definite ring of infiltration. The other rabbit developed a more diffuse infiltration in the deeper layers of the cornea.

Bacilli taken from cultures from case 3 were introduced beneath the conjunctiva of an eye of a rabbit. For the next three days the eye was moderately congested. A suspension of the bacilli was introduced into the vitreous through the ciliary body, thus traumatizing it. One day later, there was a distinct ring of infiltration around the cornea, which within three hours was not visible because the entire cornea was opaque. Panophthalmitis rapidly developed. Bacteria were also introduced into the vitreous through the sclera. The cornea became opaque, and panophthalmitis developed. A definite corneal abscess could not be seen.

The examination of eyes inoculated with cultures from case 1 showed a more complete picture of the pathologic condition of ring infiltration than could be seen in the fragments removed from our patients. The infiltration was densest near the limbus on both sides, where it extended almost through the limbus leaving a clear cornea in the center. It tended to approach the surface of the cornea at the more central border of the ring. The whole eye was involved in the diffuse infiltration of panophthalmitis.

Examination of the cornea of the rabbit inoculated with culture from case 3 suggested a more advanced stage in the development of ring abscess. There was a dense infiltration in the deeper layers of the cornea, especially at the limbus. Toward the center of the cornea, the infiltration was more marked superficially, but it extended to the center of the cornea. The remainder of the eye was densely infiltrated with a well developed panophthalmitis.

SURVEY OF THE LITERATURE

From a survey of the literature, ring abscess would seem to be a rather rare condition. Fuchs [1] reported a case seen in 1881; Giri [2] reported a case of purpura followed by panophthalmitis with ring abscess; Morax reported one following pneumonia; Herbert,[3] one from

1. Fuchs: Am. J. Ophth. **56**:1, 1903.
2. Giri: Brit. J. Ophth. **2**:611, 1918.
3. Herbert, H.: Brit. J. Ophth. **2**:187, 1919.

plague; Axenfeld,[4] one from endocarditis, and Hirschberg,[5] one from cystitis. The remainder of the cases reported resulted from injury to the eye. Twelve of these were from penetrating wounds, two as a result of burns, one from an ulcer and three following cataract extraction. The type of organism found varied greatly. Hay bacillus was the most commonly found, being present in four cases, pneumococcus in two, staphylococcus in two, *Bacillus pyocyaneus* in two, *Bacillus proteus* in one, *Bacillus fluorescens* in one, *Bacillus mycoides* in one and unidentified bacilli in the remainder.

Most of the authors reported the loss of the affected eye, but in three cases useful vision was retained.

Several theories are advanced as to the cause of the ring of infiltration found in the cornea. Fuchs [1] believed that bacteria found their way into the anterior chamber through the wound, the cornea being attacked from the inner side and the leukocytes reaching the cornea from the ciliary vessels, thus localizing the ring of infiltration a short distance from the limbus.

Some investigators have produced ring abscess experimentally. Hoppe [6] introduced hay bacillus, *Staphylococcus albus* and *Bacillus pyocyaneus* into the anterior chamber and vitreous of rabbits, with resulting ring abscess and panophthalmitis. Cramer [7] was able to produce a slight ring of infiltration in the same manner with *Staphylococcus albus*. Kodama [8] produced a ring abscess by the inoculation of the vitreous with hay bacillus. Hanke [9] caused a ring abscess by abrasion of the cornea of rabbits. Küffler [10] was able to produce ring abscess with a certain strain of hay bacillus. He found that it was necessary to injure the attachment of the iris in some way, and was successful after the complete removal of the iris, iridodialysis and cyclodialysis, if the bacilli were introduced into the vitreous.

In spite of the paucity of reported cases in the literature, it seems to us that the condition is more common than this would indicate. Many cases undoubtedly have not been reported. Perhaps some are not recognized, because the entire cornea has become opaque when first seen, or the picture of panophthalmitis has developed, which diverts attention from the appearance of the cornea. Undoubtedly, the prognosis is bad in most cases. Parsons stated that the condition almost invariably ends

4. Axenfeld: Am. J. Ophth. **40**:3, 1894.
5. Hirschberg: Ztschr. f. Augenh. **4**:188, 1880.
6. Hoppe: Klin. Monatsbl. f. Augenh. **45**:333, 1907.
7. Cramer: Klin. Monatsbl. f. Augenh. **48**:620, 1910.
8. Kodama: Klin. Monatsbl. f. Augenh. **48**:624, 1910.
9. Hanke: Ztschr. f. Augenh. **10**:373, 1903.
10. Küffler: Arch. f. Ophth. **78**:227, 1911.

in the loss of the eye. As before stated, however, in three cases reported in the literature some useful vision has been recovered.

When the patient is seen early, the use of heat, foreign protein therapy and atropine are indicated, and it would seem a logical procedure to relieve the tension on the cornea by incising it just internal to the ring, and allowing the aqueous to escape. When the central cornea has become opaque and light perception is abolished, as often occurs within

Data from the Literature

Name	No. of Cases	Causative Factor	Observations and Bacteriology	Result
Herbert[3]	1	Plague	No bacteriologic work reported	Death
Giri[3]	1	Purpura	No bacteriologic work reported	Evisceration
Abe	..	.....	Reported experimental work on rabbits, producing panophthalmitis with hay bacillus	
	1	Injury	Author thinks it a trophic lesion	
Snell: Am. J. Ophth. **4**: 419, 1921	1	Burn	No bacteriologic work reported	
Hoppe[6]	3	Injury (2); ulcer (1)	Found hay bacillus, S. albus and bacillus pyocyaneus; produced ring abscess in rabbits by inoculation of vitreous with hay bacillus	One eye ruptured; vision 20/65; other two enucleated
Cramer[7]	1	Steel injury	Found Staphylococcus albus and zerosis; inoculation into eyes of rabbit produced only a slight reaction	Evisceration
Kodama[8]	1	Corneal wound	Found hay bacillus; introduction into eyes of rabbit caused a ring abscess	?
Fuchs[1]	10	Injury postoperative	Found Staphylococcus albus, streptococci and an unidentified bacillus	One recovery of some vision
Hanke[9]	..	Burn	Found Bacillus fluorescens; produced ring abscess in rabbits	Some return of vision
Morax	..	Metastatic from pneumonia	Found pneumococci	Death
Flieringa: Klin. Monatsbl. f. Augenh. **69**: 241, 1921; Am. J. Ophth. **8**: 70, 1925	1	Injury	Found hay bacilli	
Küffler: Arch. f. Ophth. **78**: 237, 1911; Berl. klin. Wehnschr. **47**: 2273, 1910	..	.....	Produced ring abscess in rabbits by inoculation of hay bacilli into vitreous, the iris previously having been traumatized	
Axenfeld[4]	1	Pneumonic endocarditis	Found pneumococci	Death
Hirschberg[5]	1	Cystitis	.....	Death
Weiss: Klin. Monatsbl. f. Augenh. **3**: 393, 1875	1	Fracture of femur	.....	Death

thirty-six hours after the ring is first observed, evisceration or enucleation usually must be performed. In spite of the extensive involvement of the cornea, we have found that simple evisceration, with removal of the necrotic portions of the cornea, has not given rise to complications.

SUMMARY

1. Four cases of typical ring abscess of the cornea are reported.

2. All were of traumatic origin, progressed rapidly to total opacification of the cornea within from two to three days, and ended in evisceration.

3. The causative organism was *B. subtilis* in two cases and probably in a third. The frequent observation in the literature of the subtilis group as the cause of this condition offers a reminder that these organisms, usually considered saprophytes, may, when introduced into the avascular cornea or vitreous, produce destructive inflammation.

4. Attempts to reproduce the condition with cultures obtained from cases were successful in two rabbits into the vitreous of which pure cultures from cases 1 and 3 were inoculated. Inoculations into the anterior chamber were not successful.

5. In pathogenesis, the introduction of organisms into the anterior chamber or vitreous, and probably also injury to the iris root, would seem to be the most important factors, though a few cases have occurred by metastasis from distant foci of infection.

6. The condition is probably more common than the literature would indicate.

Ophthalmologic Review

OCULAR RESIDUA AND SEQUELAE OF EPIDEMIC ENCEPHALITIS *

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PHILADELPHIA

A little over ten years ago the great influenza epidemic raged over the entire world. About the same time in Europe, and shortly thereafter in America, another fearful scourge, epidemic encephalitis, made its appearance, and, in sporadic form, still exists. While not as deadly in its immediate results as was the influenza epidemic, it is perhaps equally as tragic when the thousands of mentally and physically incapacitated survivors of the disease are taken into consideration. According to Parsons,[1] about 40 per cent become disabled, "as it appears from the after-histories of some 3,000 patients."

That ocular disturbances play a prominent rôle in the symptomatology of the early stages of this disease is well known. Such eye symptoms as diplopia and paresis of accommodation are often among the earliest manifestations. In most instances, fortunately, they are transitory and disappear in a few days or weeks. In many cases, however, they persist for many months or years. In others, again there may be an interval of three or four years between the acute and the post-encephalitic or parkinsonian stage, which resembles syphilis in this regard (Folk[2]).

Frequently the ocular disorders are the chief symptoms of which the patient complains, and the oculist is the first medical attendant consulted.

The ocular disorders of epidemic encephalitis are confined chiefly to the motor nerves of the eye; the third and sixth nerves are most frequently involved, and occasionally also the trochlear. The optic nerve is rarely affected. Even in the early stages, optic neuritis and papilledema are rather infrequent occurrences, though a number of cases have been recorded. Very few, however, have been confirmed by autopsy. At a recent symposium on the subject by the sections of neurology and ophthalmology of the Royal Society of Medicine in London, the opinion was almost unanimous that papilledema and optic neuritis in epidemic encephalitis are exceedingly rare. Williamson-

* Submitted for publication, Feb. 29, 1929.

1. Parsons, Allan C.: Post-Encephalitis and Its Problems, Proc. Roy. Soc. Med. **8**:1307, 1928.

2. Folk, M. L.: Ocular Disorders in Encephalitis Lethargica, Am. J. Ophth. **9**:677, 1926.

Noble [3] mentioned four cases of papilledema in all of which the diagnosis of epidemic encephalitis was found to be incorrect at autopsy. In discussing the differential diagnosis between encephalitis and tumor of the brain, he quoted a report by the ministry of health in which the statement is made that "in the diagnosis of lethargic encephalitis, a negative sign of value is the absence of optic neuritis." In a recent case under my observation in which there was a bilateral optic neuritis with clinical manifestations of epidemic encephalitis, the diagnosis at autopsy proved to be a glioma of the frontal lobe.

Paralysis of the external ocular muscles is a frequent occurrence. Cords and Blank [4] found it in 28 per cent of their fifty postencephalitic cases. Bielchowsky [5] reported a case in which one and one-half years after acute encephalitis there was a bilateral oculomotor palsy. According to Economo,[6] the paralysis is always bilateral. Jaensch [7] is not in accord with this view. In his sixty-eight postencephalitic cases, he found abducens paralysis in four, paralysis of the sixth nerve in one eye and of the third nerve in the other eye in one, unilateral paralysis of the internal rectus in four, unilateral paralysis of the oculomotor in six, and bilateral paralysis of the oculomotor with involvement of all its external branches in two cases. In one case there was an isolated trochlear paresis. The same author related the case of a young girl under observation for three years after symptoms of acute encephalitis had subsided. She suffered from attacks of diplopia. Repeated examinations revealed a recurrent right external and internal ophthalmoplegia. During the height of the attack there was complete ptosis which partly receded in a few days; afterward she complained of diplopia. At first the attacks occurred every three or four weeks. In the last two years, however, the intervals increased to six months.

According to Collier,[8] ophthalmoplegia in epidemic encephalitis is met with in two forms, peripheral and nuclear. In the peripheral form, recovery always takes place. He defined nuclear ophthalmoplegia as an irregular ophthalmoplegia hardly ever complete, affecting both eyes and always leaving the optic axes unparallel. It may be transient when occurring in slight degree, but in the more severe degrees, after clearing up to some extent, it always leaves some permanent paralysis. The

3. Williamson-Noble: Proc. Roy. Soc. Med. **21**:985, 1928.

4. Cords, R., and Blank, I.: Okuläre Restsymptome nach Encephalitis epidemica, Klin. Monatsbl. f. Augenh. **72**:394, 1924.

5. Bielchowsky, A.: Die Augensymptome bei der Encephalitis epidemica, Klin. Wchnschr. **4**:120, 1925.

6. Economo: Abstr. in Zentralbl. f. d. ges. Ophth. **3**:442, 1920.

7. Jaensch, P. A.: Spät und Restsymptome an den Augen bei Encephalitis epidemica, Klin. Monatsbl. f. Augenh. **77**:813, 1926.

8. Collier, J.: Proc. Roy. Soc. Med. **21**:985, 1928.

lesion may involve any part of the nuclear mass and therefore produce every sort of defect in the movements of the two eyes. In epidemic encephalitis Collier has never seen paralysis of any conjugate movement with the optic axes remaining parallel, and no diplopia. He believed the explanation is that this disease spares the white matter to a large extent, and consequently true conjugate paralysis (which is essentially a tract defect) does not occur.

Bramwell [9] mentioned two definite signs which he observed and which, in his opinion, occur in almost no other disease. One is the loss of upward movements of the eyes and the other is a curious oscillation of the eyeballs when the patient attempts to perform certain movements of the eyes.

Reese [10] reported a case of paralysis of downward movement and of convergence with paresis of accommodation.

Williamson-Noble referred to oculogyric crises which consist of spasmodic attacks of conjugate deviation of the eyes, most frequently vertical and often associated with a marked emotional factor. It is apt to be regarded as a manifestation of hysteria. Pardee,[11] Barkas,[12] Hohman,[13] Lehrfeld,[14] Fischer [15] and others have written on this condition of curious tonic eye-fits or "Blickkrampf" as the Germans call it. It occurs most frequently, if not exclusively, in the parkinsonian syndrome of epidemic encephalitis. Muskens [16] localized the lesion just in front of the posterior commissure, and ascribed the sign to partial interference with the supravestibular centers and their paleocortical connections. One of the reasons for localizing the lesion just in front of the posterior commissure is the frequent association of tonic lateral position of the eyes with the upward or downward position; the two bundles involved in these two forms of forced movement are close together only at one point, namely, just in front of the posterior commissure.

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9. Bramwell, E.: Proc. Roy. Soc. Med. **21**:985, 1928.

10. Reese, W. S.: Postencephalitic Paralysis of Downward Movement and of Convergence, Atlantic M. J. **30**:423, 1927.

11. Pardee, J.: Paroxysmal Oculogyric Crises in Parkinsonian Encephalitis, Am. J. M. Sc. **175**:683, 1928.

12. Barkas, M. R.: Tonic Spasm of the Eyes in Conjugate Deviation, Lancet **2**:330, 1926.

13. Hohman, Leslie B.: Forced Conjugate Upward Movements of the Eyes in Postencephalitic Parkinson's Syndrome, J. A. M. A. **84**:1489 (May 16) 1925.

14. Lehrfeld, L.: Ocular Findings in Postencephalitic Syndrome, Am. J. Ophth. **9**:272, 1926.

15. Fischer, M.: Der extrapyramidale Blickkrampf als postencephalitisches Symptom, Arch. f. Psychiat. u. Nervenh. **77**:303, 1926.

16. Muskens, L. J. J.: Ocular Pathology of Postencephalitic Disturbance in Light of Recent Anatomical and Physiological Research, J. Neurol. & Psychopath. **8**:132, 1927-1928.

According to Young,[17] blepharospasm is a common symptom in the parkinsonian syndrome of postencephalitic origin. He found it in 92 per cent of his thirty-nine cases.

Another symptom of the eyes in the postencephalitic parkinsonian stage is a peculiar rigid condition of the eyeballs to which Nonne [18] first called attention in 1919. Later Cords [19] systematically investigated and described it, and coined the expression, "myostatic rigidity of the eyes." The face has a masklike appearance, the action of the facial muscles appears to be entirely abolished, and there is not a trace of mimicry in the face. The eyes are expressionless and stare vacantly into space or at some fixed object for many minutes and even hours at a time. Stellwag's sign is also present. In some of the cases in which Jaensch observed this eye symptom, winking took place from every 50 to 100 seconds. It is not a question of paralysis of the eye muscles in these cases. When the patient is aroused, the eyes can be moved with an effort, but they soon assume the same fixed position.

Ptosis, which is a frequent occurrence in the acute stage, has also been reported as a residual symptom by Cords and Blank,[4] Hess,[20] Goldflamm,[21] Kassner [22] and others.

Nystagmus or nystagmoid jerkings has been frequently observed as a late symptom. It is of greatest diagnostic value when the nystagmus is up or down, while a lateral nystagmus is present in numerous other diseases.

Convergence paralysis, partial or total, is held by many to be one of the most frequent eye symptoms observed in the parkinsonian syndrome. According to Williamson-Noble, it is often associated with slight irregularity of the pupils, poor reaction to light and little or no pupil reaction on attempting accommodation-convergence. Cords and Blank found weakness of convergence in 66 per cent of their cases. The importance of being able to differentiate between congenital and acquired convergence paresis is emphasized by them. They remind one that insufficiency of convergence is not an infrequent occurrence, and

17. Young, A. W.: A Clinical Analysis of an Extrapyramidal Syndrome; Paralysis Agitans and Postencephalitic Parkinsonism, J. Neurol. & Psychopath. **8**:9, 1927-1928.

18. Nonne, M.: Zum Kapitel des epidemisch auftretenden Bulbärmyelitis und Encephalitis des Hirnstammes, Deutsche Ztschr. f. Nervenh. **64**:185, 1919.

19. Cords, R.: Die Myostatiche Starre der Augen, Klin. Monatsbl. f. Augenh. **66**:1, 1921.

20. Hess, F. O.: Die Folgezustände der akuten Encephalitis epidemica, München. med. Wchnschr. **68**:481, 1921.

21. Goldflamm, S.: Die grosse Encephalitis-epidemie des Jahres 1920, Deutsche Ztschr. f. Nervenh. **73**:1, 1922.

22. Kassner, Hans: Augenkomplikationen als Spätfolgen von Encephalitis lethargica, Klin. Monatsbl. f. Augenh. **72**:59, 1924.

that the convergence act is often absent in one-sided amblyopia, in high myopia and in cases in which there is an absence of binocular vision.

Paresis of accommodaion, less frequently a complete paralysis, which is one of the earliest eye symptoms in epidemic encephalitis, is a frequent residual symptom. It has also been observed as a late complication. Reys [23] saw it in a patient two and one-half years after the acute illness. M. Myer observed it in seven of his sixty late cases, and Cords and Blank reported it present in 26 per cent of their postencephalitic cases.

Mehrtens and Barkan [24] studied the pupils and measured the power of accommodation in thirty-six subacute and chronic cases. They found an internal ophthalmoplegia in 44 per cent and a reduction of the power of accommodation as an isolated sign in 36 per cent, or a total of 80 per cent, "thus making the paresis of the ciliary muscle the outstanding ocular lesion in epidemic encephalitis." A paresis of accommodation was formerly regarded only as a complication in diphtheria. Henceforth one must also think of encephalitis.

Collier, in speaking of the relation between herpes and encephalitis, related that during a period of epidemic encephalitis he saw an unusual number of cases of unilateral total internal ophthalmoplegia in patients who were not syphilitic. Some were in young persons just past the age of puberty. He thought that these instances might have been due to a herpetic lesion of the ciliary ganglion. None of these cases, however, showed any other signs of epidemic encephalitis.

The pupil has always been regarded as a valuable aid in the diagnosis of nervous disorders. In the study of neurology one is taught to associate certain pupillary phenomena with certain pathologic conditions. The various changes concerning the size, form, reflexes and reactions of the pupil in epidemic encephalitis, however, have tended to weaken this doctrine somewhat, and have taught one to be more cautious in drawing conclusions. Hess,[25] Westphal [26] and others called attention to the extraordinary inconstancy of the pupil in this disease. Changes varying from normal to iridoplegia may be observed during the course of the illness, and in some cases surprising changes in size and reaction were noted in a single day. Among the residual symptoms, anisocoria has been reported by many observers. Miosis, frequently seen in the acute stage, is believed to be due to a cramplike condition of the pupillary sphincter. This is a transient symptom.

23. Reys, L.: La clinique ophthalmologie, quoted by Cords and Blank: Klin. Monatsbl. f. Augenh. **72**:394, 1924.

24. Mehrtens and Barkan: Researches of the Pupillary Reactions in Epidemic Encephalitis, Arch. Neurol. & Psychiat. **10**:399 (Oct.) 1923.

25. Hess, F. O.: Ueber fortlaufende Beobachtungen bei unseren Enzephalitiskranken, München. med. Wchnschr. **71**:6, 1924.

26. Westphal, A.: Wechselnde Pupillenstarre, Deutsche med. Wchnschr. **51**:2101, 1925.

Diminished or absent convergence reaction with normal light reflex is considered by Adie [27] and others to be of great diagnostic value in epidemic encephalitis. It has been shown, however, that in these cases there is also a weakness of convergence, and the failure of the pupil to react is secondary to the convergence paralysis.

The question whether the Argyll Robertson pupil occurs as a late or a residual symptom in epidemic encephalitis is of special importance. According to teachings, the Argyll Robertson pupils are pathognomonic of neurosyphilis. Nonne, Economo and others have reported the Argyll Robertson pupil in epidemic encephalitis. Foster Kennedy [28] said: "We have been misleading students by teaching that the Argyll Robertson pupil is a sign of syphilis only. It can result from toxic infective or mechanical attack on the oculomotor nuclei." Edmund Adler [29] and others pointed out that the Argyll Robertson pupil in epidemic encephalitis is a passing symptom and disappears rapidly, while in syphilis it persists. Cords and Blank reported the absence of the Argyll Robertson sign in all of their fifty postencephalitic cases. Mehrtens and Barkan studied the pupil with the von Hess pupilloscope in their thirty-six postencephalitic cases. They did not find the true Argyll Robertson pupil in a single instance. They called attention to the importance of measuring the power of accommodation in every case. Otherwise, an ophthalmoplegia interna might be erroneously diagnosed as Argyll Robertson pupil. They also stated: "The pupil in encephalitis is often mydriatic and is round or ovoid, in marked contrast to the serrated irregularity of the syphilitic pupil."

SUMMARY

1. A review of the literature reveals that ocular complications are a frequent occurrence in the postencephalitic state.

2. Residual and late eye symptoms are confined chiefly to the outer and inner musculature of the eye.

3. Vertical nystagmus, forced conjugate upward movements and oculogyric crises are valuable signs in the diagnosis of the so-called parkinsonian stage of encephalitis.

4. Paresis of accommodation, which was formerly regarded as a complication of diphtheria, is a frequent residual symptom in encephalitis.

5. The Argyll Robertson pupil has been frequently observed in acute encephalitis, but it is of rare occurrence in chronic encephalitis.

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27. Adie: Proc. Roy. Soc. Med. **21**:985, 1928.

28. Kennedy, Foster: Abstr. of discussion on Mehrtens and Barkan, Arch. Neurol. & Psychiat. **10**:399 (Oct.) 1923.

29. Adler, Edmund: Zum Verlaufe der Encephalitis epidemica (und über einige ungewöhnliche Erscheinungsformen derselben), Med. Klin. **18**:1142, 1922.

Notes and News

EDITED BY DR. GEORGE S. DERBY

SOCIETY NEWS

Colorado Congress of Ophthalmology and Oto-Laryngology and Summer Graduate Course.—The seventh annual summer graduate course and the Colorado Congress of Ophthalmology and Oto-Laryngology will be held this year from June 17 to 29, inclusive. The last two days, June 28 and 29, will be given over to the congress for the presentation of papers. It will be noted that the time selected is one month earlier than is customary, and immediately precedes important meetings to be held in the West during July. These are: the Pacific Coast Oto-Ophthalmological Society at Salt Lake City, July 1 to 3; The American Laryngological, Rhinological and Otological Society at San Francisco, July 4 and 5; and the American Medical Association at Portland, July 8 to 12. For those who anticipate attending any or all of these meetings, the time set for the graduate course and Colorado congress will be especially convenient.

Illustrated lectures will be given daily from 10 to 12 a. m. and from 2 to 4 p. m. The following physicians will speak on the subjects given: Glaucoma and Its Treatment, E. C. Ellett, Memphis, Tenn.; Fields of Vision in Brain Disease, Clifford Walker, Los Angeles; Fundus Photography, Arthur J. Bedell, Albany, N. Y.; Practical Applications of Physiological Optics, Edward Jackson, Denver; Ophthalmoscopic Findings in General Diseases, Henry P. Wagener, Rochester, Minn.; Histopathology of Fundus Changes that are Seen with the Ophthalmoscope, William C. Finnoff, Denver.

Demonstrations will be given between the hours of 8 and 10 a. m. daily in the various hospitals throughout the city and in the assembly hall. The following subjects will be covered: Cross Cylinder in Determining the Strength and Axis of Cylinders in Refraction; Methods of Examination in Refraction; Methods of Taking the Visual Fields; Histopathology of Ocular Changes in General Diseases; Testing Anomalies of the Ocular Muscles. The physicians demonstrating will be: William H. Crisp, Donald H. O'Rourke, John A. McCaw, James M. Shields and William M. Bane.

The size of the class for the summer graduate course is limited to sixty-five members and will be divided into small sections for the demonstrations and clinics. All ethical practitioners are eligible for the course, and their applications, when accompanied by a check for $50, will be accepted in the order in which they are received. These should be mailed to Dr. H. L. Whitaker, secretary-treasurer, Republic Building, Denver. Further information also may be obtained from Dr. Whitaker.

The congress, which will be held the last two days, is open to all who may wish to attend, without charge.

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom was held on April 11, 12 and 13. The following program was presented, Mr. Charles Howard Usher presiding:

On Thursday, April 11, papers were read by Mr. E. Treacher Collins on "Formative Fibrous Tissue Reaction in the Eye"; Mr. F. A. Williamson Noble on "Graded Squint Operations"; Mr. M. L. Hepburn on "Some Unsolved Problems in Connection with Diseases of the Choroid," and Mr. Clark Soutar on "Uveo-Parotid Fever." A discussion on "Heterophoria" was opened by Messrs. E.

Maddox, C. Goulden and Group Captain C. Clements. Others taking part in the discussion were Messrs. Affleck Greeves, Ernest Clarke, H. Grimsdale and H. Neame. The annual dinner took place at the Langham Hotel.

On Friday, April 12, papers were read by Mr. Duke-Elder on "The Nature of the Vitreous"; Mr. Davenport on "The Nature of Tay's Choroiditis and Allied Conditions"; Miss Ida Mann on "Demonstrations of the Phylogenetic Value of the Lower Retinal Field"; Mr. G. F. Alexander on "Suggestions on the Instrumentation of Cataract Operations" and "The Great Antiquity of Removal of Cataract from the Eye," and Mr. E. Wolff on "A Note on the Pathology of Orbital Hemorrhage and Inflammation." A clinical meeting took place at the Royal Westminster Ophthalmic Hospital. A discussion on "The Diagnosis and Treatment of Ocular Tuberculosis" was opened by Sir Arnold Lawson, Dr. R. A. Young and Mr. S. Browning; Messrs. A. L. Whitehead, W. S. Duke-Elder and others also took part.

On Saturday, April 13, papers were read by Mr. M. H. Whiting on "Optic Atrophy Following Hemorrhage from the Alimentary Tract"; Mr. B. Cridland on "The Prevention of Minor Eye Injuries in Industry" (an exhibit of various goggles and other appliances to protect the eyes from injury was held in the museum), and Mr. F. T. Ridley on "Gas Gangrene Panophthalmitis."

SPECIAL TRAIN TO PACIFIC COAST DURING JULY

A special train will be run to accommodate the ophthalmologists and otolaryngologists attending the meetings on the Pacific Coast during July.

The train will leave Chicago on Sunday, June 30, arriving in Salt Lake City, Tuesday, July 2, allowing attendance at the Pacific Coast Oto-Ophthalmological meeting that day. The train will leave Salt Lake City Tuesday night, arriving in San Francisco Wednesday night, July 3.

The party will stay in San Francisco, Thursday, Friday and Saturday for the Triological meeting, leaving for Portland Saturday afternoon, reaching there Sunday afternoon, July 7.

The special train is being sponsored by the American Laryngological, Rhinological and Otological Society and the Pacific Coast Oto-Ophthalmological Society. The details are being handled by the Union Pacific Railroad.

Any one wishing special information, should address the Union Pacific Railroad, General Passenger Department, Omaha, or Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha, who has been asked to represent the societies interested.

Richmond Ophthalmological and Oto-Laryngological Society.—Dr. Emmette T. Gatewood has been elected president of the Richmond Ophthalmological and Oto-Laryngological Society for the ensuing year.

Chicago Ophthalmological Society.—Drs. Harry W. Woodruff, Joliet, Ill., and William F. Moncreiff discussed strabismus before the Chicago Ophthalmological Society, March 18.

UNIVERSITY NEWS

Dr. Joseph E. White has retired from the professorship of ophthalmology in the Medical College of Virginia and has been appointed emeritus professor. Dr. Emory Hill becomes professor of ophthalmology.

GENERAL

Owing to the death of A. Darier in 1927 and that of G. Gerard recently, it has been necessary to suspend the publication of *La clinique ophthalmologique* in 1929. However, publication will probably be renewed in 1930.

Obituary

LEWIS H. TAYLOR, M.D.
1850-1928

Lewis H. Taylor died at Wilkes-Barre, Pa., on Nov. 5, 1928, following a cardiac embolism. He was born on July 29, 1850, at Taylorsville, now Washington's Crossing, Bucks County, Pa. He was the son of Samuel B. and Margaret H. (Baker) Taylor. His ancestors were of old Quaker stock who were among the settlers of the present site of Tacony, Pa., in the latter part of the seventeenth century.

After graduating from the State Normal School at Millersville, Lancaster County, in July, 1871, he taught school for six years in Wilkes-Barre, being principal of a grammar school and later of the high school. He received the degree of Doctor of Medicine from the University of Pennsylvania in 1880. Dr. Taylor observed the practice throughout his life of preserving letters and records, and these early evidences of the admiration and respect engendered by him among the influential people of Wilkes-Barre foretold, even in that early day, the unusual success and happiness he was to develop in a busy professional life of forty-eight years. After graduation he spent several months in Philadelphia practicing ophthalmology and otolaryngology in association with the late Dr. Samuel D. Risley.

He returned to Wilkes-Barre and opened his office for general practice. When he went to Vienna in 1883, he had achieved an unusual degree of success in his work. His exceptional personality, the exemplary habits of his private life, and his energy and intelligence, early won for him valuable connections with older men. This interest and helpfulness he later returned many times over to the men younger than himself as he became the older practitioner, consultant, and finally, dean of the medical profession in Wilkes-Barre.

He spent the winter of 1883 and 1884 in Vienna studying ophthalmology and otolaryngology, and on his return to Wilkes-Barre, limited himself to these specialties, becoming one of the earliest, if not the first, specialist in northeastern Pennsylvania. At this time he married Emily Beard Hollenback, by whom he is survived, with one daughter, Anna.

His rise in the medical profession was gradual and uninterrupted. From 1885 to 1894, he was medical inspector of the Pennsylvania State Board of Health, and in this capacity studied and reported on the epidemic of typhoid fever in Plymouth. He identified the source of infection, and his report, written in his own beautiful style, portrays as interesting a bit of detective work as can be found in many works of

fiction. In his study of a subsequent typhoid epidemic, he again demonstrated the danger to the city by contamination of the water supply. By his insistence on preventive hygienic measures, he brought on himself the criticism and enmity of men of influence, which only a character such as his own could ignore.

He was president of the Luzerne County Medical Society in 1885, of the Pennsylvania State Medical Society in 1912 and chairman of the Section on Ophthalmology of the American Medical Association in 1906. He had previously been secretary of the Section, and it was during his tenure of this office that the presessional volume of the transactions was first published. It is my impression that Dr. Taylor originated, or at least shared materially in the conception of the plan of the presessional volume and carried forward the idea against the discouraging opposition of the usual quota of doubting Thomases who said it could not be done.

He was a member of the Lehigh Valley Medical Association, of the Philadelphia Pathological Society, of the Medical Club of Philadelphia, of the American Academy of Ophthalmology and Oto-Laryngology, of the American Ophthalmological Society, of the American Otological Society and of the American College of Surgeons. These were not empty honors to him. He was a regular and frequent attendant at the meetings of all of them, besides being a member of several international congresses. Probably no man in his section of the country had so wide an acquaintance among the members of the medical profession of the nation as did Dr. Taylor. Attendance at meetings was a duty and a pleasure to him. Deafness was no deterrent, but the perfection in recent years of mechanical aids to the deaf made his enjoyment of the program more satisfactory to him. To a limited circle, he was frank in his criticism of a poor speaker, but always kind. He himself developed an extraordinarily pleasing delivery, and this coupled with a marvelous memory made him a welcome speaker on any occasion. In his earlier years, he had read numerous papers of technical and professional interest. In his later years, most of them concerned medical libraries and medical history.

Dr. Taylor was the first resident physician to be appointed for duty at the Wilkes-Barre General Hospital; he was ophthalmologist on the staff, and later president of the board of directors. He was the first secretary of the staff and served in this capacity for years. He was also a trustee of the First Methodist Church, of the Y. M. C. A., of the Wyoming Historical and Geological Society, and of the Wyoming Seminary, and president of the trustees of the Osterhout Free Library. In 1891, he received the degree of Master of Arts from Lafayette College.

Probably the outstanding monument of his professional life is the library of the Luzerne County Medical Society at Wilkes-Barre, which he established by his foresight and enthusiasm about thirty-five years ago. He had been the librarian from the time of its founding until his death. The last afternoon before he was taken ill, he spent at the library with his beloved books. By his own efforts, the nucleus was formed by gift and loan of books and periodicals. It was largely by reason of the growth of the library that the Luzerne County Medical Society erected a fireproof building to house the collection. Through Dr. Taylor's efforts, the first sum for endowment was secured from the late John Welles Hollenback. Under Dr. Taylor's guidance, the library grew to over 8,000 books and bound periodicals, and by his own legacy the endowment was increased to $34,000.

He was a frequent visitor to medical libraries throughout the country, and had long been a member of the American Library Association. When he went to Minneapolis last June, part of the purpose of his trip was to visit the library of the Mayo Clinic, to visit the librarian and to look for duplicate copies. It was through this insatiable interest that he found time during a busy professional life to create, rear and endow for the future, a splendid medical library that can well serve as a model for the smaller cities.

In his later years, circumstances made him one of the executors of the estate of John Welles Hollenback. His professional activities were accordingly curtailed, but his energies were applied to a wider world of business activity. With this came a deeper appreciation by his community at large. Even in this altered sphere, his innate honesty and sincerity were not changed. One business associate has remarked that Dr. Taylor's fine sense of fairness would not allow him to take advantage over a weaker person in a business decision, even though the question of honesty or legality could not be raised. The end of his earthly activities came as he would have wished could he have decreed it. He was taken ill immediately after ushering out the last patient for the day.

Dr. Taylor was generous to a fault. He knew no jealousy or envy, and stood by his own efforts and his own goodness. He was the friend and counselor of physicians, especially in their youth. His was a life sweet and full, replete with respect for God and cleanliness for his fellowman, desiring riches only for the means of happiness, and then usually for that of others. He leaves a void scarcely to be filled, but a memory sweet and lasting.

LEWIS T. BUCKMAN.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Physiology

IRIS MOVEMENTS IN BLIND MICE. CLYDE E. KEELER, Am. J. Physiol. **81**:107, 1927.

Measurements of the pupillary light reflexes in a pure strain of mice which never develop rods in the retina showed that the iris of the mouse, under photostimulation, dilates and contracts independently of the functional capacity of the retina. (The author had previously satisfied himself from a detailed series of experiments with behavior that these animals were completely devoid of vision.) A longer time than in the normal animal was required in these rodless mice for the response of the iris to light of the same intensity; the actual degree of contraction was less than normal.

The author believes that these experiments prove that in mammals the iris may function independently of vision.

F. H. ADLER.

THE FACTORS CONTROLLING THE INTRA-OCULAR PRESSURE. W. S. D. ELDER, Proc. Roy. Soc. Med. **21**:37, 1928.

This is a review of the author's conclusions regarding the maintenance and control of intra-ocular pressure. During the past few years the author has sought for a satisfactory answer to the question whether the aqueous humor is a secretion and, as such, the product of work done by the ciliary epithelium, or whether it is a dialysate from blood serum. If the latter is correct, no other forces are necessary than those known chemical and physical forces afforded by the capillary blood pressure and the osmotic pressure of the blood acting across a semipermeable membrane. The author believes that the latter hypothesis is correct, based on the following facts which he has been able to verify:

The chemical composition of aqueous, its protein content, the distribution of electrolytes and nonelectrolytes, and its osmotic pressure as compared with that of blood serum, all fit in with this theory. If aqueous humor is a dialysate, it must have a definite composition in order to be in equilibrium with its parent fluid and this composition the author has shown it to possess. Further, the peculiar disposition of ions between blood and aqueous would necessitate a certain difference in osmotic pressure between the two. The author's measurements of the osmotic pressure of the blood and aqueous show that this relationship also holds good.

Further he has presented evidence which suggests that the hydrostatic pressure in the capillaries is sufficient to equilibrate this osmotic pressure and to act in the formation of a protein-free filtrate such as aqueous represents. The unequal distribution of ions in blood and aqueous necessitates a certain difference in electrical potential between the two. This difference in potential the author has found actually exists. Finally the hydrogen ion concentration in the blood has been shown to be greater than that of aqueous which is also a necessary postulate of such a system. Since the aqueous, therefore, appears to be in complete equilibrium, chemical, osmotic, hydrostatic and electrostatic, with the capillary blood and since it shows all the complex properties of a dialysate, the author concludes that it seems logical to assume it as such in the absence of proof to the contrary. Normal intra-ocular pressure, therefore, is maintained at the

equilibrium level by the intracapillary pressure on the one hand and the difference between the osmotic pressure of the blood plasma and that of the aqueous humor on the other. Variations in these two factors will lead to marked changes in intra-ocular pressure, as the author has shown in numerous and varied experiments. Then follows a discussion of the paper, mainly by Treacher Collins and Mr. Mayou. The article is a clear exposition and review of Elder's work, leaving out the experimental details which are sometimes confusing to those unfamiliar with laboratory methods.

F. H. Adler.

Vision

Visual Acuity Within the Area Centralis and Its Relation to Eye Movements and Fixation. Frank W. Weymouth, Don Carlos Hines, Lawrence H. Acres, John E. Raaf and Maynard C. Wheeler, Am. J. Ophth. **11**:947 (Dec.) 1928.

With the Ives visual acuity test carefully arranged to eliminate objectionable factors, examinations were made on three eyes to determine the visual acuity within the area centralis. For each eye the acuity was determined at the center and for eight tangent areas on each side, in the horizontal and vertical meridians. A field of about 2 degrees, 50 minutes was covered. In one subject, measurement was possible at an angular distance of 10.64 minutes from fixation. The experimental procedure is described in detail.

The results of the tests prove that the area centralis shows a uniform sensory gradient similar to that for the entire retina; the gradient is continuous from a sharp maximum at the center of the fovea, a decided difference being found, as close as 0.0528 mm.; the different meridians show different rates of change, greatest in the temporal, less in the nasal, still less in the inferior and least in the superior meridian. The authors support the view that the sensory gradient is the basic factor in movements and fixation of the eye.

There are three tables, seven graphs and a bibliography.

A. Cowan.

A Factor in the Visual Inferiority of the Extra-Foveal Retina. G. Ovio, Ann. di ottal. e clin. ocul. **56**:1057 (Dec.) 1928.

When the eye is focused on a point, rays from this point focus only at the macula. Oblique rays reaching other parts of the retina are not in focus and cause circles of diffusion the size and disturbing effect of which are calculated by the author. He believes that the anatomic specialization of the macula for distinct vision may have developed secondarily in that region where rays focus when the eye is fixed on an object. Thus, while in birds with lateral eyes the fovea is placed at the posterior pole, in those such as owls with some binocular vision, foveae are present at the temporal side, and in aquatic animals such as the cephalopods, foveae are present in the upper part of the retina to aid in looking down. In parts of the retina which do not habitually receive rays in focus, such close approximation of the retinal elements would be of no value and therefore is not developed.

S. R. Gifford.

Methods of Examination

Application of the Bar-Reader to Campimetry, Stereocampimetry, and Other Purposes. Morris Davidson, Am. J. Ophth. **11**:966 (Dec.) 1928.

The author places a test object on the bar and utilizes its crossed physiologic doubling and projection on the plane of fixation. As a test card he uses dead-

black cardboard 14 by 24 cm. One side is ruled in squares for campimetry at 25 cm. It has three white, round fixation objects attached, one in the center of the card and one in the center of each outlined blind spot. On the reverse side is arranged a strip of Jaeger type, a row of three red fixation objects, a row of three green fixation objects and a row of three small, round-headed pins, a black one in the center and a white one on each side. The bar is a dead-black piece of cardboard 4 by 14 cm.

The bar held vertically between the eyes and the test card not only hides from one eye a part that is seen by the other eye, but it is itself an object of perception. The following tests are described: rapid campimetry with binocular fixation; detection of simulation; tests of fusion and stereopsis and training, and determination of near points of accommodation and convergence and deviations for near points.

A. COWAN.

A SENSITIVE TEST FOR EQUALITY OF ACCOMMODATION. J. I. PASCAL, Am. J. Ophth. **12**:29 (Jan.) 1929.

The author makes a retinoscopic examination of the patient and compares the shadow movement of each eye as he gradually approaches the patient with the retinoscope. The patient fixes a mirror, called the "dynascope," above the retinoscopic mirror which reflects a series of small charts placed on the lower part of the retinoscope. The examiner flashes the light rapidly, first in one eye then in the other, moving toward the patient and taking care to keep in the median line while he continues to shadow both eyes. If the accommodation of both eyes is equal, the "with" movement will change at the same time in each eye. The quality of accommodation can also be tested by this method by noting the sustaining power of each eye by means of the shadows.

W. S. REESE.

Experimental Pathology

EXPERIMENTAL TUBERCULOSIS OF THE CORNEA. L. CARRÈRE. Ann. d'ocul. **165**:853, 1928.

To approach clinical conditions as near as possible, Carrère injected small quantities of Koch's bacilli under the conjunctiva, into the anterior chamber and behind the iris of guinea-pigs and dogs. In the guinea-pigs, all the subconjunctival injections with one exception produced local lesions of little importance. This one exception, presented on about the eighteenth day a slight elevation at the limbus, resembling a phyctenule. On the twentieth day, two points of infiltration of the center of the cornea were noted; these later coalesced, while the remainder of the cornea was hazy. At this point the animal was killed.

An injection was made at the limbus in a dog. The cornea developed a localized infiltration that disappeared in a few days. Seventeen days after the inoculation, a small spot appeared in the center of the cornea; this gradually spread, infiltrating almost the entire stroma. Biopsy revealed the same cellular contents as were present in the former experiment; namely, lymphocytes in a state of degeneration, polymorphonuclear leukocytes, neutrophils and masses of disorganized cells together with a small number of tubercle bacilli.

Next, inoculations were made into the anterior and posterior chambers, by withdrawing a small amount of aqueous into a syringe in which finely pulverized tubercle bacilli had been placed, and reinjecting the liquid.

Injections into the anterior chamber caused no immediate reaction. In about twelve days, however, a slight pericorneal injection appeared, together with a pupillary exudate. Later, after three or four weeks, whitish-yellow nodules appeared on the iris; these increased little by little until the whole iris was invaded. The cornea became involved at the same time.

The inoculation behind the iris caused a somewhat different reaction. In fifteen days, a slight pericorneal injection appeared without any lesion of the iris. Ten days later, or on the twenty-fifth day, a small lump appeared at the lower angle of the iris, together with a marked hyperemia of the adjacent conjunctiva. On the thirtieth day the lower part of the middle layers of the cornea, near the limbus, appeared infiltrated. Later this area spread involving the entire cornea, except the deepest part of the stroma. A descemetitis, however, was produced. During this period the iris nodule increased, extending over to the posterior surface of the cornea below, while the remainder of the iris was free from involvement.

Pathologic examination revealed invasion of the iridocorneal angle, the spaces of Fontana, the canal of Schlemm, stroma of the cornea and iris, and descemets membrane with histiocytes, polymorphonuclear leukocytes, lymphocytes and plasmocytes.

H. W. SCARLETT.

EXPERIMENTS ON THE ORIGIN OF TRAUMATIC MYDRIASIS. FUKUMI ISHIKAWA, Arch. f. Ophth. **120**:317, 1928.

The application of blunt force to a rabbit's eye never caused a persistent mydriasis but, for the most part, a transitory myosis. When the pupil had been contracted by eserine, however, the same degree of violence led to a mydriasis similar in all respects to the traumatic mydriasis seen in man.

The author states that the origin of a traumatic mydriasis in rabbits, as well as in man, is chiefly due to damage to the sphincter muscles. Histologic sections showed distinct changes in the sphincter muscle immediately after trauma.

F. H. ADLER.

SOME EXPERIMENTAL RESEARCHES ON CORNEAL VESSELS. HOLGER AHLERS, Acta ophth. **5**:99, 1927.

After traumatizing the cornea of rabbits, Ahlers studied the vascular invasion of the layers of the tissue. He found that the initial vascular invasion of the superficial or deep tissue is the formation of long slender blind capillaries which he calls sprouts. The superficial vessels which are anterior to the substantia propia send their sprouts irregularly in toward the center. When two sprouts meet, they form a loop from which other sprouts may emerge, giving the classic arborization structure. Arterioles and venules run separate courses, while in the substantia propia of the cornea the afferent and efferent vessels follow the same direction. In contradistinction to superficial vascular invasion the deeper vascular sprouts are more regular in the course, forming either straight lines or slightly curved lines of which two or more generally run strictly parallel and anastomose in pointed loops giving the typical brush or broom structure. The more superficial vessels show more branching than the deeper ones. The normal network in the rabbit corresponds closely to that of the human eye. The vessels everywhere end in closed loops, inward toward the cornea and there are no free sprouts. The superficial vessels are thicker and more numerous than the deeper scleral vessels.

A. M. YUDKIN.

Lids

PALPEBRAL LUPUS TREATED WITH SUCCESS BY EXCISION AND A FREE GRAFT. L. DUPUY-DUTEMPS, Ann. d'ocul. **165**:849, 1928.

According to Dupuy-Detemps, the treatment of lupus by free grafts from a part of the body not subject to this disease has fallen into greater disuse than it deserves. The method is simple and quick.

For small lesions the simple excision of the area is sufficient, undermining the edges of the skin and bringing the lips together with two or three sutures. For more extensive lesions, however, involving half the lid or more, it is necessary to cover the excised lesion with a cutaneous graft.

It is a question of importance from what part of the body the graft is taken. A pedicle flap from the face for example will show invasion by lupus after a variable time, while a graft transported from the trunk or the thigh will remain free from involvement throughout its entire extent, although it may be encircled by the disease. The author thinks that this is due to the fact that free grafts, taken from parts unaccustomed to invasion by tuberculosis, retain their natural resistance.

A case of lupus of almost the entire left upper lid, involving the eyebrow and extending for a short distance over the forehead, was presented in detail. The patient underwent two operations. At the first one, the entire skin of the upper lid, up to the superior orbital ridge, was excised and covered with a dermo-epidermic graft from the thigh. Two months later the remaining portion of the lesion was removed, and the area covered by a graft from the thigh. A blepharorrhaphy was performed before the first operation, and freed one month after the second operation.

Two years after the last operative intervention there was no sign of recurrence. Pathologic examination of the excised tissue showed it to be typical tuberculous lupus.

H. W. SCARLETT.

Lacrimal Apparatus

DIAGNOSIS AND TREATMENT OF DACRYOCYSTITIS. L. DAILY and R. K. DAILY, Am. J. Ophth. **12**:1 (Jan.) 1929.

A plea is made for control of cases of lacrimal sac by ophthalmologists, the rhinologist being used as a surgical consultant. Diagnosis by means of fluorescein, irrigation and roentgenogram are discussed. The authors now use iodized oil 40 per cent for roentgen purposes because it is always ready, is more fluid and easier to inject. Bismuth paste is still used when x-rays cannot be made of the patient immediately after injection. Pictures are taken in the occipitofrontal and lateral positions. The bismuth is irrigated away, this irrigation not being necessary when iodized oil is used. Several illustrative pictures are shown.

Before resorting to operation, Besredkas bacterial filtrates are tried. These are prepared by growing pneumococci and streptococci on 1.5 per cent dextrose bouillon from eight to fourteen days. The solution is sedimented and the clear portion injected into the sac. If this is ineffectual, the Halle-West operation of dacryocystorhinostomy is done. This method is described after first reviewing the anatomy of the lacrimal fossa. Three cases are reported.

W. S. REESE.

Conjunctiva

NEVUS OF THE BULBAR CONJUNCTIVA. F. H. RODIN and E. M. HALL, Am. J. Ophth. **12**:24 (Jan.) 1929.

In the case of a boy, aged 6, who was first seen in August, 1927, the mother a year previously had noticed a small brown spot on the white of the right eye, which had increased in size and recently had become surrounded by a red zone. Examination disclosed an irregular brown area about 2 by 3 mm. on the bulbar conjunctiva of the right eye, 7 mm. from the limbus. It was surrounded by a hyperemic zone about 2 mm. wide; it was not elevated and was freely movable. The growth was removed, and showed no signs of recurrence a year later. The histologic diagnosis was: a pigmented nevus of the conjunctiva.

The twelve cases reported in the literature during 1925 and 1926 are summarized; the tendency of these growths to become malignant is emphasized.

W. S. REESE.

A CASE OF RHINOSPORIDIUM KINEALYI. J. N. DUGGAN, Brit. J. Ophth. **12**:526 (Oct.) 1928.

The ocular type of *Rhinosporidium kinealyi* is rare. The spore was first discovered in a nasal polyp by Seeber of Buenos Aires. The author's patient was a Mohammedan, aged 52. The growth began as a red papule, six months before examination. In the right eye a papilloma-like growth, the free end of which was covered by a rounded and thick scab, protruded from the palpebral fissure at its inner end. It arose from the semilunar fold and bulbar conjunctiva adjacent to it by a broad base which extended from just external to the inner canthus, to within 4 or 5 mm. of the inner limbus. In width it extended from 5 mm. above the horizontal line to the fornix. It was dark red and fairly vascular. The lymphatics were not enlarged. The growth was removed and found not to involve the sclerotica.

Characteristics of the growth are that it affects stratified epithelium, and forms cysts of varying size lined by flat epithelium; the cysts burst, discharging spores, after they have grown. The cyst is then invaded by leukocytes. Microscopically, there is a fair amount of fibrous and fibrocellular tissue. The cysts are situated in the subepithelial layer. Some contain granular material, while others are empty. It grows on Sabourard's medium. Inoculations into other animals are negative. The author says that it is strange that the growth should be found in a Mohammedan, as was noticed by Elliot and Kirkpatrick. Photomicrographs and references are included.

W. ZENTMAYER.

MYCOSIS OF THE PALPEBRAL CONJUNCTIVA. G. SCALZITTI, Ann. di ottal. e clin. ocul. **56**:1023 (Nov.) 1928.

A boy, aged 11, showed a marked swelling of one upper lid which had been present for six months. Granulations had been excised three times but had recurred each time. Two yellow nodules, the size of a pinhead, were present just inside the border of the lid, surrounded by numerous smaller white points in an area of infiltrated conjunctiva. Wassermann and Pirquet reactions were negative. The globe was not affected. A portion of the conjunctiva, removed for section, showed a nodule resembling a tubercle with threads staining with hematoxylin resembling those of a fungus. A fragment inoculated in the anterior chamber of a rabbit produced a small nodule of the iris which was still present after five months. Ten injections of iodized oil caused the lesions to clear up entirely. The type of

fungus could not be determined from the sections, and cultures evidently were not made. The clinical resemblance to Pereyra's case of streptothricosis of the conjunctiva is noted. A bibliography, a photograph and photomicrographs are included.

S. R. GIFFORD.

Lens

THE USE OF LENS PROTEIN TESTS BEFORE CATARACT OPERATIONS. R. H. COURTNEY, Am. J. Ophth. **12**:20 (Jan.) 1929.

The author tested preoperatively, twenty-six patients with cataract for sensitivity to lens protein; all were normal. Twelve patients were tested postoperatively, five of whom showed a pronounced sensitivity to lens protein. No definite relation between the amount of unabsorbed cortex and the development of sensitization was noted, nor was there any difference between healing in the sensitive and nonsensitive cases. Five cases are reported, the last being complicated by a uveitis. The author urged the lens protein test before any operation on a soft cataract, particularly a needling, and especially, before operating on the second eye.

W. S. REESE.

BLOOD PRESSURE AND CATARACT. L. S. BATTISTA, Ann. di ottal. e clin. ocul. **56**:961, 1928.

The relation of the arterial blood pressure to intra-ocular tension is discussed, the author concluding that no constant relation between the two has been proved to exist. So far as retinal arteriosclerosis is related to cataract, while the two frequently co-exist, advanced cases of retinal arteriosclerosis may exist with clear lenses, and vice-versa.

The author examined 300 patients between 60 and 95 years of age in a hospital for the aged and infirm. Of this number, 143, or 48 per cent, showed opacities of the lens. The percentage of those affected at various ages was: from 60 to 75 years; 50 per cent; from 75 to 80, 75 per cent; from 85 to 90, 90 per cent, and from 90 to 95, 100 per cent. Between the ages of 70 and 75 cortical opacities were frequent, five patients showing mature cataracts, while beyond 85 years of age nuclear sclerosis was most often found, the cortex being for the most part clear. Before the age of 70, the opacity occurred only in one eye, while beyond that age it was always bilateral. Three diabetic patients were found in the series, all having bilateral cortical cataract.

Sixty-three patients had a blood pressure below 130 mm. of mercury; in 147 patients, it was from 145 to 170, and in 69 patients, more than 170. Fifty-seven per cent of the patients with opacities of the lens showed normal blood pressure (below 130), while of the sixty-nine patients with hypertension (over 170) thirty-nine showed clear lenses and thirty opacities of the lens. Those with blood pressure from 130 to 170 showed opacities of the lens in 72 per cent. Thus no relation could be observed between cataract and blood pressure, nor was any relation found between intra-ocular tension and blood pressure. Hence, whatever hypothetic substance in the blood stream may be supposed to cause arteriosclerosis has apparently no such deleterious effect on the lens, and the author is forced to consider cataract as a senile involution such as is seen in other ectodermal structures. A table of cases and an extensive bibliography are included.

S. R. GIFFORD.

THE RELATION BETWEEN CONGENITAL CATARACT AND CHANGES IN THE NEURO-VEGETATIVE APPARATUS. C. GIANUANTONI, Ann. di ottal. e clin. ocul. **56**:1063 (Dec.) 1928.

The importance of proper equilibrium between the sympathetic and parasympathetic nervous systems in the development of the organism is discussed. Any disturbance of this balance during development, or in that of the mother during fetal life may give rise to anomalies in the growth of various organs. The author investigated the neurovegetative system of twelve cases of congenital cataract. Pharmacodynamic tests were performed, inoculations of 5 per cent morphia and 1 per cent epinephrine being made according to the Pirquet technic. The reflexes giving information about the vegetative nervous system, such as the oculocardiac reflex and the appearance of dermographia, were recorded, and the blood calcium was determined.

In none of the cases was any evidence found of endocrine dysfunction. Seven cases showed by their reflexes a hyperfunction of the parasympathetic system, while five showed no predominance of either system. The Wassermann reaction was negative in all patients. The blood picture and blood calcium in all cases were within normal limits; roentgenograms of the sella showed no marked abnormalities. The skin tests with morphia and epinephrine varied without showing any constant relation to the other observations. No evidence of rachitis or tetany was found. Thus the only evidence that vegetative dysfunction plays a part in causing congenital cataract was the evidence of vagotonia in seven cases, the importance of which, the author concludes, will depend on further observations in a large group of cases. A bibliography is included.

S. R. GIFFORD.

CATARACT, TETANY AND DWARFISM. P. SAINTON and G. RENAUD, Arch. d'opht. **45**:391 (June) 1928.

The picture of tetanic cataract, as Weill and Nordmann have described it in their research on cataract and its relations to general pathology, is classic.

The authors call attention to the cases already reported and the experimental evidence which has been obtained.

They then report the case of a patient, aged 47, 128 cm. in height and weighing 38 Kg. He had first lost the sight of his right eye from cataract which was operated on with a good visual result. Although his left eye failed later in a similar manner he came under observation because of symptoms of tetany. The cataract in this left eye was so far advanced that it was difficult to determine its variety.

He had been subject to seasonal attacks, which occurred usually from January to March, since he was 12 years of age. He had the usual clinical signs of tetany. X-ray pictures demonstrated changes in the long bones somewhat like those found in rickets.

It seems probable that in this case disease of the parathyroid glands is chiefly responsible for the whole clinical picture.

S. B. MARLOW.

ON THE OXIDATION MECHANISM OF THE CRYSTALLINE LENS. GUNNAR AHLGREN, Acta ophth. **5**:1, 1927.

Ahlgren investigated the methylene blue metabolism of the lens tissue by a method introduced by Thunberg in the study of other tissue. He noted that the oxido-reduction power of the lens is considerably diminished on exposure to a

temperature of —80 F. and that it is merely abated when subjected to a temperature of +60 F. Because of these changes in the oxido-reduction power of the lens he believes that the tissue has an oxidation enzyme of the type called dehydrogenosis which plays an important part in the oxidation mechanism of the lens. Dehydrogenosis affecting the oxidation of fumaric, malic, glycero-phosphoric, lactic, citric, acetic and the amino-acids — alanine and valine — has been found to exist in the lens. In a similar test, however, succino-dehydrogenase, which has been found in all other tissues, is absent in the lenticular mass. To a certain extent, therefore, the metabolism in the lens follows a different path from that of muscle and other tissue. The lens cortex shows a greater oxidation intensity than the lens nucleus. Ahlgren also noted that the oxidation in the lens cortex of some thyroidectomized rabbits is increased from the subnormal intensity by the administration of suitable concentrations of thyroxin. High concentrations of thyroxin retard the oxidation intensity of normal and thyroprive lens cortex.

A. M. YUDKIN.

THE QUESTION OF ENDOCRINE ETIOLOGY OF JUVENILE CATARACTS. V. GRÖNHOLM, Acta ophth. **5**:166, 1927.

From an analysis of five cases of bilateral juvenile cataracts which were accompanied by some manifestation of disturbed internal secretion and a review of the literature on the subject of cataract formation in endocrine disturbance, Grönholm classifies the cases into the following groups: In the first group he places the lenticular opacities which are accompanied by disturbed internal secretion, such as diabetic cataract due to hypofunction of the pancreas; tetany cataract produced by hypofunction of the parathyroid glands; myotony cataract seen often in hypofunction of the thyroid and parathyroid glands; acromegaly cataract due to hyperactivity of the anterior lobe of the hypophysis; diabetes insipidus cataract produced by hyperfunction of the pars intermedia of the hypophysis, and pluriglandular cataract due to a dysfunction of internal secretion. In the second group are the cases which show no signs of disturbance of the internal secreting glands. These include cataracts which are produced by the introduction of toxic substance into the ocular tissue through the blood stream. If the toxin disturbs the internal secreting gland, and these in turn the lens, the cases are to be classified in the first group.

A. M. YUDKIN.

Uvea

FREE CYST IN THE ANTERIOR CHAMBER. C. A. CLAPP, Am. J. Ophth. **11**:862, 1928.

The author has found but seventeen other similar cases reported. The family and antecedent history of the patient are of little importance. The cysts are usually singular; in one case, however, there were seven cysts and in only one case was the condition bilateral. The cysts may occur at any period of life but are more often seen in young persons.

Several theories have been advanced to explain them: (1) They are the products of developmental arrest and possibly associated with pupillary membranes. (2) They are produced by excrescences of pigment epithelium at the pupillary margin or perhaps from the posterior surface of the iris. (3) They develop from embryonal retinal tissue from the pars ciliaris. (4) Their content is vitreous. The latter view was suggested by Greeves, who studied Spicer's case microscopically. Meller found a delicate connective tissue wall with numerous pigment granules on the outer surface.

The author's case was seen in a man, aged 21. In the anterior chamber of the right eye there was a freely movable oval body measuring 0.75 by 1 mm. Its normal position was in the region of 7 o'clock; if the patient was placed prone, however, the cyst immediately fell to the center of the cornea. The biomicroscope showed the surface of the cyst to be uneven and covered by pigment granules on a delicate basement membrane. The interior was filled with a clear substance. No pupillary membrane was present. A brief summary of the cases reported since 1912 is given. A colored drawing and bibliography are included.

W. ZENTMAYER.

CHANGES IN THE PERIPHERY OF THE FUNDUS IN MYOPIA AND IN SENILE EYES. KARL REHSTEINER, Arch. f. Ophth. **120**:282, 1928.

The author has examined 304 eyes ophthalmoscopically after maximal dilatation of the pupil with homatropine and cocaine. The indirect method was used. In order that the extreme periphery of the fundus could be seen, the patient was requested to turn his eyes ad maximum in all different positions; a condensing lens of 13 diopters was so held in front of the eye that a prismatic effect was obtained. The refraction was measured by ophthalmometry and by skiascopy. The frequency of changes in the fundus increased with increasing myopia as the accompanying table demonstrates.

Changes in the Fundus in Relation to Myopia

Extent of Myopia, Diopters	Peripheral Lesions, Per Cent
1 to 10	26
11 to 20	50
20 +	75

In a number of cases of anisometropia the lesions were found only in the myopic eye. These lesions consisted of pigmented spots for the most part in front of the retinal vessels and atrophic areas. With increasing age of the patients the frequency of these myopic changes in the periphery of the fundus increased. In elderly people quite frequently, similar lesions were found in both eyes regardless of the extent of the myopia. The type of degeneration in both the myopic and the senile eyes was the same, consisting of white or yellowish lesions of different size, containing proliferated pigment and forming long bands by confluence.

The author believes that these lesions are similar to the degenerations found in the posterior segment of the globe.

F. H. ADLER.

Glaucoma

FURTHER INVESTIGATIONS INTO THE RESULTS OF IRIDENCLEISIS OPERATIONS BY HOLTH'S METHOD, PARTICULARLY AS REGARDS THE METHOD COMBINED WITH MERIDIONAL IRIDOTOMY. H. G. A. GJESSING, Acta ophth. **5**:149, 1927.

A review of his work on 113 eyes operated on for glaucoma by the Holth method, Gjessing notes that the relief from tension not infrequently comes about slowly and therefore requires the use of a miotic until six months after the operation. None of the cases showed a positive Seidel fluorescein test; 83 per cent maintained normal tension without the further use of miotics, and in no case was the tension subnormal. In approximately 83 per cent of the cases the vision was unaltered or increased. The field of vision was preserved in 76 per cent, and in four cases the field was considerably increased.

He concludes that the iridencleisis of Holth, especially that combined with meridional iridotomy gives a relatively certain and lasting result without exposing the eye to any considerable later risk because when the operation is properly performed, no thin bleb is formed over the included iris. This iris will be covered by relatively thick conjunctiva, which will give a negative Seidel phenomenon. The ciliary body will not be exposed to the extent that is frequently the case in the trephining operation of Elliot. Both these factors tend to prevent late infection. Hypotension is not found. The cosmetic results are good because none of the fistula-forming material is lost, the pupil is but slightly dislocated and dazzling of the eye is thus avoided.

A. M. YUDKIN.

Neurology

FREQUENCY OF THE BERNARD-HORNER SYNDROME. H. W. SCARLETT, Am. J. Ophth. **11**:961 (Dec.) 1928.

Sixteen cases, observed during a period of four years, were studied in detail and analyzed. A review of the literature was made, and the anatomy of the cervical sympathetic nerve fibers, together with their cerebral connections, as at present understood, was given.

Enophthalmos, ptosis and narrowing of the palpebral fissure were encountered in all the cases reported, while miosis was absent in only one case. Heterochromia irides was seen twice in the series, while hypotonus was present six times. Among the associated signs, hemiatrophy of the face and unilateral flushing were observed in fourteen cases. The near point of the affected eye was less in every patient, the average difference being 3 cm. In four instances, vision in the eye showing the syndrome was less than that in the other eye.

The etiology of the condition was known in one case, that being syringomyelia. This patient showed the typical Klumpke type of paralysis of the inner side of the forearm and the small muscles of the hand, as well as the most complete and pronounced ocular pupillary symptoms. The presence of a mother and son in the series suggests the possibility of heredity as an etiologic factor.

The conclusions were: (1) Paralysis of the cervical sympathetic nerve, in full or in part, is more frequent than the literature indicates. (2) The cause of such paralysis was determined in only one case of this series. (3) The difference between the near points of the two eyes was greater in those patients showing the most complete syndrome. (4) The average difference between the pupils before the instillation of cocaine was 1.8 mm., while afterward it was 3 mm. (5) The width of the palpebral fissure averaged 3.5 mm. less on the affected side.

AUTHOR'S SUMMARY.

General Diseases

LIGHT TRANSMISSION BY COLORED SPECTACLE LENSES IN THE VISIBLE SPECTRUM. LAURANCE D. REDWAY, Am. J. Ophth. **11**:973 (Dec.) 1928.

An examination of the various makes of colored spectacle lenses is made to ascertain the characteristics and the quality of light transmitted by each; a table of the transmission of blue, green, red and white light through each of thirty-two varieties of lens is given.

In general, lenses transmitting blue will increase the resolving power of the eye for near vision, providing there is sufficient illumination. Lenses transmitting a great deal of green give increased brilliancy to objects seen by the wearer, and

are comfortable and well tolerated. Glasses transmitting orange or red are desirable for detailed distant vision and exaggerated contrast.

It seems only common sense that lens shall be utilized which most closely approximates the requirements of individual use. It is possible to use ordinary gelatin filter material between thin wafers of glass and mounted in a frame, thus affording a great variety of filter effects not otherwise obtainable. A table of the transmission of blue, green, yellow, red and white light for nineteen Wratten light filters is given.

A. COWAN.

GYNECOLOGY AND DISEASES OF THE EYE. E. VOGT, Monatschr. f. Geburtsh. u. Gynäk. **80**:1, 1928; abstr., J. A. M. A. **91**:1933 (Dec. 15) 1928.

The onset of the menarche may exert a favorable influence on inflammatory and hemorrhagic diseases of the eye. Vogt explains this on the assumption that menstruation rids the body of certain toxins (menotoxins). In the presence of disease of the eye it may be desirable to hasten the appearance of the first menstruation. Suppression of the menstruation sometimes has a bad effect on diseases of the eye. Albuminuric retinitis occurs in nephropathy of pregnancy as well as in true chronic nephritis and nephrosis. It is most frequent in multiparas and appears usually after the sixth or seventh month. If it appears in the first six months, it is an indication for the interruption of the pregnancy. When it appears later, opinion as to the proper procedure is divided. Vogt believes it better to wait and interrupt the pregnancy only if the eye lesion becomes worse. If after the evacuation of the uterus the retinitis does not entirely clear up, future pregnancies should be avoided. Eclamptic amaurosis in itself need cause no particular concern. The pregnancy should be interrupted immediately in nearly all cases of optic neuritis. Only in exceptional cases is expectant treatment permissible, under close observation, till the child is viable. Future pregnancies must be avoided. Operation on the eye during pregnancy leads to abortion in at most a third of the cases, but it is always advisable when operating on a pregnant woman to avoid the time at which the menstrual period would have fallen if she had not been pregnant, since at this time the uterus is more sensitive. Hemorrhages into the eye during labor need not cause anxiety, even when copious. The menopause sometimes has a bad effect on diseases of the eye, perhaps because of increased blood pressure, perhaps because of retention of menotoxins. The possible effect of a coexisting lesion of the eye should be taken into account when the establishment of an artificial menopause is under consideration.

W. ZENTMAYER.

ON ERYTHROPIA AND XANTHOPSIA IN HEMERALOPIA. EDMUND CARSTEN, Acta ophth. **5**:88, 1927.

With a special apparatus designed for determining the ocular sensibility to red, green and violet, Carsten examined a patient suffering from deprivation hemeralopia (vitamin A deficiency). He noted that violet perception on the whole was impaired; perception of red and green was relatively stronger and perception of violet relatively less than normal. She therefore perceived a mixture of red and green as yellow. A normal person would see other shades because of the stronger impression of violet. He ascribed the erythropia to deficient adaptation, or lack of power to increase the sensibility for violet. In his opinion, the experiments published by Tscherning, showing that dark vision actually is violet vision, corroborated his observation that hemeralopia and reduced sensibility for short-waved rays were coincident.

A. M. YUDKIN.

Operations

SURGICAL TECHNIQUE FOR THE REMOVAL OF SUBRETINAL CYSTICERCUS. R. SILVA, Am. J. Ophth. **2**:869 (Nov.) 1928.

Three cases are reported. In two of them the parasite was subretinal, and in the third it was in the vitreous. In the first case there was a large subretinal cysticercus in the region of the macula, reaching to the optic nerve. The vision had been lost for about one year from complete detachment of the retina. Under local anesthesia the external rectus was clamped and severed leaving a stump attached to the sclera. A meridional incision was made down to the choroid, 16 mm. from the corneal limbus: the herniated choroid was then incised, the envelop was opened and a cysticercus, 14 mm. in length, expelled. The sclera, muscle and conjunctiva were then sutured. No reaction followed. One year later the eyeball was intact.

In the second case there was evidence of the parasite having migrated from the region of the macula to the inferior external quadrant of the eye. The parasite was exuded through a small meridional incision. Normal central vision was retained.

In the third case surgical intervention for the removal of the parasite was refused. Subsequently the eye was removed because of violent iridocyclitis. A dead cysticercus was found in the vitreous.

The author gives the diagnostic symptoms and the methods used for localization of the parasite. He points out that the prognosis depends not only on the location but on the timeliness of surgical intervention. Even when operative success is assured, the final result depends on the alteration sustained by the eye prior to the intervention. The Krönlein operation need not be performed.

W. ZENTMAYER.

EXTRACTION OF INTRA-OCULAR FOREIGN BODIES WITH THE MELLINGER MAGNET. E. LEONARDI, Ann. di ottal. e clin. ocul. **56**:1096 (Dec.) 1928.

The author reports the cases of twenty-one extractions with the inner pole or ring magnet. He states that this magnet, which is little used in Italy, possesses certain advantages over the giant magnets of Haab and others. In using it, the patient's head is placed in a solanoid, the eye being near the center. The current produces an intense magnetic field inside the solanoid, so that a metallic foreign body inside it also gives off a magnetic field. If an electrode of soft iron is placed in the solanoid, it also produces a magnetic field which, on account of its size, is stronger than that of the foreign body and attracts the latter strongly.

Since the foreign body is itself a magnet, and both it and the electrode give off parallel lines of force, the attraction of the electrode is almost the same at any point inside the solanoid, and does not decrease so markedly with distance as does that of the giant magnets. An even more important advantage is the ease with which the electrodes can be manipulated, as compared with the giant magnets. The attraction of the magnet may be varied by using electrodes of different sizes; some of these may be inserted conveniently in a corneal or scleral wound.

Of eight cases in which the scleral route was used, two resulted in phthisis bulbi and enucleation, three retained useful vision and three very good vision. The results by the corneal route in thirteen cases were better, two eyes being lost, three retaining useful, and five very good, vision. In spite of this slight difference in results, the author believes that the corneal route is safer when the lens is

intact. In no case was the iris torn by the extraction, nor did the foreign body ever become imbedded in the ciliary body; no other complications attributable to the method developed. A bibliography is included.

S. R. GIFFORD.

ON THE TECHNIQUE OF OPENING THE SHALLOW ANTERIOR CHAMBER. A. ELSCHNIG, Klin. Monatsbl. f. Augenh. **80**:382, 1928.

The usual procedure of using a Graefe knife in performing a peripheral iridectomy for glaucoma when the anterior chamber is shallow has certain disadvantages; for example, the section must be made at an acute angle so that the iris cannot be drawn out completely to the root. Following the technic of Gayet, Elschnig advises this procedure:

Anesthesia is accomplished by instillations of cocaine and the retrobulbar injection of 1 cc. of 2 per cent procaine hydrochloride with the addition of epinephrine 1:10. As a rule, a suture is placed through the superior rectus muscle. An incision is made through the conjunctiva, from 4 to 5 mm. from the limbus, for about one third of the total corneal circumference; it is made with a scalpel and the conjunctiva is dissected down to the cornea with scissors. With a scalpel, an incision from 6 to 7 mm. long is made through the limbus by careful dissection into the anterior chamber. Bleeding is partially prevented by the slow and careful dissection with the scalpel and with pledgets of epinephrine. Pregl's solution may also be used. The aqueous is allowed to flow out slowly. The wound is then enlarged to the desired size, the sphincter seized with forceps and the iris drawn down toward the center of the pupil and then up and out through the wound. The iridectomy can then be made in the usual manner.

In case of synechia at the root of the iris, the root may first be freed with a spatula. The conjunctiva is placed in position and the eye closed. The advantages of this operation are:

1. Avoidance of hemorrhage.
2. Slow evacuation of the anterior chamber avoiding intra-ocular hemorrhage and loss of vitreous by bursting of the zonula.
3. The iris remains uninjured from the incision.
4. There is no danger, therefore, of wounding the lens. Even if the scalpel does go too deep the zonula and not the lens would be injured.
5. The excision of the iris is total, that is, up to its root.

This operation is recommended also for the performance of optical iridectomy in the presence of a shallow anterior chamber.

F. H. ADLER.

Therapeutics

GENERAL LIGHT BATH TREATMENT OF SOME DISEASES OF THE EYE. C. F. BENTZEN, Acta ophth. **5**:29, 1927.

After seven years' observation with regard to heliotherapy, Bentzen concludes that it is not a specific therapeutic measure in all ocular ailments. Some constitutional diseases of the eye which have resisted other treatment have manifested favorable results with this therapy. He believes the ocular improvement is due to the beneficial influence of the light baths on the general health. This therapeutic measure is indicated in iridocyclitis, particularly the tuberculous form. Persons more than 50 years of age should not be subjected to this treatment. Severe cases of phlyctenular keratoconjunctivitis react well to heliotherapy, but hospital treatment, he notes, is the best cure in the majority of these cases. Tuberculous scleritis and recurrent vitreous hemorrhages do not yield to treatment with light.

A. M. YUDKIN.

Comparative Ophthalmology

THE ANATOMY AND NEURO-PHYSIOLOGY OF THE CAT'S NICTITATING MEMBRANE. H. ZERNIK, Arch. f. d. ges. Physiol. **220**:593, 1928.

Stimulation of the spinal cord in five cats showed the fibers for retraction of the nictitating membrane confined to the region from the eighth cervical to the second thoracic segments. The fibers run through the stellate ganglion in the cervical sympathetic nerve and form a synapse in the superior cervical ganglion. From there the nerves run directly to the bulla ossea, then through this and vanish in the bone on the floor of the skull.

Their further course into the nictitating membrane could not be followed. The muscles of the nictitating membrane consist of two sharply differentiated groups of fibers, that is, those in the long axis, which cause a retraction of the membrane, and those situated transversely, which protrude it.

F. H. ADLER.

Pharmacology

MICROCHEMICAL AND HISTOCHEMICAL INVESTIGATION OF MERCURY IN THE EYE AND INTRA-OCULAR FLUIDS. E. REGANATI, Ann. di ottal. e clin. ocul. **56**:1114 (Dec.) 1928.

Tissues were left from twelve to twenty-four hours in an acidified 25 per cent solution of stannic chloride, which first converts mercuric salts to mercurous salts and then reduces these to metallic mercury; this can be seen microscopically after it is embedded in paraffin. For the intra-ocular fluids, the same solution was added to the fluid in equal amounts, and the precipitate examined microscopically. Albino rabbits were used. One group was given toxic doses of mercuric chloride subcutaneously, causing death on the fourth or fifth day; a second group was given doses of the same drug corresponding by weight to therapeutic doses for man, while a third group was given doses of mild mercurous chloride larger than the therapeutic dosage. In the first group, mercury was found in the aqueous after twenty-four hours and in the vitreous after twenty hours. In the ocular tissues no mercury was found, one hour after the injection. After one day a few granules of mercury were seen in the deeper layers of the cornea and sclera and in the subconjunctival tissue. After thirty-six hours mercury was found constantly in these tissues and also around the optic nerves. Just before death after four or five days, all the intra-ocular structures except the lens contained mercury.

In the second group of animals which were given therapeutic doses, mercury was found in the aqueous, and in some animals also in the vitreous, retina, optic nerve and subconjunctival tissue.

None of the animals given mild mercurous chloride showed mercury in the eye or intra-ocular fluids.

The author concludes that the efficacy of parenteral administration for specific ocular lesions is demonstrated, and believes it to be superior to the painful method of subconjunctival injection. A bibliography is included.

S. R. GIFFORD.

EXPERIMENTS ON IONTOPHORESIS. K. STEINDORFF, Arch. f. Ophth. **120**:175, 1928.

The eyes of rabbits were anesthetized with holocaine and electrodes placed on the cornea and on the back of the neck. Quantitative estimations of the iodine

content of the aqueous, vitreous and lens were made by means of the Heffter-Anten method (*Archiv für experimentelle Pathologie und Pharmakologie* **48**:331, 1902). Thirty minutes' exposure to a current of 10 milliamperes showed that a considerable amount of iodine had entered the eyes. The amount of iodine increased with the increasing strength of current. Sixty minutes later, the iodine which had entered the anterior chamber by a two minute exposure to current of 2 milliamperes had disappeared. Stronger currents introduce just as much iodine into the vitreous as into the aqueous. Iodine never penetrates the lens by means of iontophoresis.

F. H. ADLER.

CORRECTION

"RETINAL DEGENERATION IN MACULAR REGION WITHOUT CEREBRAL SYMPTOMS," BY DR. ARNOLD KNAPP

Through oversight in the editorial offices, the following bibliography was omitted from the paper by Dr. Arnold Knapp, "Retinal Degeneration in Macular Region Without Cerebral Symptoms," which appeared in the last (March) issue of the ARCHIVES OF OPHTHALMOLOGY:

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Society Transactions

EDITED BY DR. GEORGE S. DERBY

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY

THOMAS H. JOHNSON, M.D., *Secretary*

Regular Meeting, Nov. 21, 1928

BERNARD SAMUELS, M.D., *Chairman*

GLASS EMBEDDED IN THE EYE. DR. MARTIN COHEN.

For twenty-eight years, glass has been embedded in the eye of a man, aged 43, without producing symptoms. He has always been in excellent health, and his physical condition shows no abnormality. The history of the injury is as follows:

On Jan. 17, 1900, while a student at the Clinton High School on East 13th Street, he was performing an experiment requiring the mixture of potassium nitrate with sulphuric acid in a retort; when the mixture was heated over a Bunsen burner, the boiling started an explosion which blew out all the window-panes in the laboratory, and threw him and others to the floor. As soon as the fumes cleared the patient perceived that he had lost the sight of the right eye. His instructor at once applied cold water to the eye and took him to Dr. Knapp's Hospital on West 12th Street, where Dr. Born examined him within an hour after the accident. Dr. Born advised the boy's relatives in regard to the treatment and the possible outcome, and at once sent the patient to the operating room.

The eye presented an incised gaping wound of the conjunctiva and sclera, about half an inch in length and situated temporally and horizontally about 4 mm. from the corneal limbus; the vitreous protruded. The patient was placed on a reclining chair and prepared for the operation. No cocaine anesthesia was used on account of the danger of infection.

After excising the prolapsed vitreous, three episcleral sutures were employed to unite the sclera and conjunctiva. No reaction resulted, and two or three days later the sutures were removed. As the vitreous body was filled with blood, no fundus examination could be made. After being in the hospital for two and a half weeks, the patient was discharged, and advised to return for future observation.

After several months the blood in the vitreous was absorbed; Dr. Born was then able to observe a distinct parallel line of a reflecting surface, about the same length as the incised scleral wound. This appearance was recognized as a piece of glass which had entered the globe at a tangent angle at the time of the explosion. The glass was embedded in the retina and underlying tissue, and was located peripherally in the upper and outer quadrant of the fundus. It was surrounded with blood and pigment, but did not protrude visibly into the vitreous. I do not know of any pathologic condition that simulates the appearance of glass.

This patient has been under the observation of Dr. Born, intermittently, for the past twenty-eight years. The vision, field and media in this eye have remained normal, without the development of any lesion. No scotoma can be demonstrated, as the foreign body is situated too peripherally. The patient has been told to

seek advice in case any irritation of the eye occurs from displacement of the foreign body.

Cases of a similar nature have been reported in the literature, proving that the globe can tolerate glass if no infection or marked irritation occurs. This inert effect of glass in the globe was due to the fact that it remained sterile as it entered and remained in the globe, and also that glass remains chemically inert under such conditions.

AN ORBITAL GROWTH. DR. MARTIN COHEN.

A woman, aged 65, who was first examined by me at the Manhattan Eye and Ear Hospital on Nov. 6, 1928, complained of slight pain and protrusion of the right eye. She gave the following history:

Sixteen years before presentation, the family, for the first time, noticed a slight bulging of the right eye which increased slowly until six years before, when it increased more rapidly. The bulging of the skin below the right orbital ridge has become more evident within the past year. During these sixteen years the patient consulted several ophthalmologists at the various clinics who, after examination and observation for varying periods, advised an operation, which she always refused. Fourteen years before presentation she consulted Dr. Marple at the New York Eye and Ear Infirmary, where she came intermittently for three years. At that time Dr. Marple referred her to the New York Hospital, where the surgeon advised an operation which she refused. Later, Dr. Marple presented her at a meeting of the Section on Ophthalmology of the Academy. At that meeting the nature of the growth could not be definitely determined. In 1920, the patient remained for ten days at the Memorial Hospital for treatment with radium, resulting in no apparent improvement. There, also, she was advised to have an operation, which she refused. After that time she sought no advice until her visit to the Manhattan Eye and Ear Hospital in 1928.

A complete physical examination at that time—including Wassermann test, a differential blood count and a roentgen examination—revealed nothing that could throw any light on the nature of the growth. No cerebral symptoms were ever complained of by the patient. No mass could be felt in any portion of the body. I have the impression that in this case there is a primary nonmalignant or a noncongenital orbital growth whose true nature cannot be determined until a microscopic examination is made after its removal.

The left eye has a vision of 20/30 and is normal in every respect. The right or involved eye has a vision of 20/100 which cannot be improved. This eye protrudes beyond the orbital margin in a straight direction, thereby stretching the optic nerve and the remaining orbital structures. The upper lid is protruded, owing to an orbital growth which also extends beyond the upper and outer orbital margin. This growth is attached to the middle portion of the roof of the orbit and extends laterally, encircling the globe above, temporally, and continues posteriorly. The growth has a doughy consistence; it is not cystic, is slightly movable and does not increase in size on bending or coughing. The motility of the eye is limited upward and outward, and, slightly, inward and downward. The ocular conjunctiva is injected. The field of vision, the tension and the pupillary reaction are normal. The cornea shows a vascular infiltration in the lower quadrant. Examination of the fundus shows the optic disk to be definitely pale and surrounded by a choroidal atrophy. Some choroidal vessels show a sclerosis. Otherwise, the fundus is normal.

I have advised operation, to which the patient has now consented, owing to the increased ocular pain and marked proptosis. I shall attempt to extirpate the

growth and preserve the eyeball by making a long conjunctival incision and shall then attempt to separate the mass with blunt dissection from the surrounding tissues. The removed growth will then be submitted for microscopic study and future report.

TRAUMATIC PULSATING EXOPHTHALMOS. DR. MARTIN COHEN.

On Aug. 31, 1928, a man, aged 21, was thrown from an automobile to the sidewalk, striking the right side of his head. He was at once transferred to the Tuxedo Memorial Hospital in an unconscious condition. On admission he was bleeding from both ears and nose. He remained irrational for four days and then complained of severe headaches, continuous loud noises in the ears and head, and pain in the left eye. There was also a loss of sensibility in the left side of the face, with facial paralysis. After a week in the hospital, exophthalmos became apparent. The patient remained in the hospital for four weeks, and then returned home to be treated by his family physician.

He was first seen by me at the Manhattan Eye and Ear Hospital on Nov. 6, 1928. Examination showed his physical condition to be normal, excluding the forementioned lesions. The chief complaints were irritability of the left eye and loud constant noises in the head and ears of the patient, which at times disturbed his sleep. These head noises could be controlled partially or completely by marked digital pressure over the carotid artery.

The right eye was normal. The left eye was proptosed straight foward; the lids and ocular conjunctiva showed venous hyperemia; the lids could not be closed completely. The movement of the eyeball upward and outward was limited. The tension, field and pupillary reaction were normal. The cornea was infiltrated in its lower quadrant; the media otherwise were transparent. The fundus showed solely a retinal venous congestion. Vision equaled 20/30. Auscultation with the stethoscope placed over the eyeball and the area between the eyebrows elicited a bruit, which disappeared on marked digital compression.

A roentgen examination of the skull by Dr. Law revealed mainly a fissured basal fracture; no fracture was observed in the neighborhood of the left cavernous sinus. The cause of traumatic pulsating exophthalmos, is stated in autopsy reports as usually due to a spicule of bone penetrating the internal carotid. The arterial blood is allowed thus to enter the cavernous sinus, causing the bruit, and the venous hyperemia of the ophthalmic vein and its branches in the orbit produces the exophthalmos.

Although the lesion occurred two and a half months before presentation, I am attempting to check the bleeding into the cavernous sinus by compression over the carotid artery with an apparatus which I suggested to a mechanic, namely, the making of a cervical truss simulating digital compression. It is possible that the pressure exerted by the truss can be applied more uniformly and maintained longer than by digital compression. This procedure is at present purely experimental; similar methods have previously been tried.

In using the truss, the patient places it over the proper area, which he determines by hearing the bruit diminished or obliterated. He then lies flat on his back and leaves the truss in position from about thirty to forty minutes, depending on the general symptoms. The patient has done this every two or three hours, excepting at night, for the past three days, with slight improvement in the condition judging from the exophthalmometric reading. I shall continue this treatment for a few more days; if more definite improvement does not become apparent, I shall advise ligation of the internal carotid artery, which is the surgical procedure now generally performed in this condition. Ligation of the artery has resulted, in a few instances,

in disastrous effects on the brain by interfering with the cerebral circulation. I am therefore attempting this compression over the carotid artery before advising operation.

DISCUSSION

DR. RAYMOND DODD: In regard to the second case of Dr. Cohen's, that of pulsating exophthalmos, just five years ago, I had two such cases within about a month. One of them was a traumatic condition resulting from an automobile accident somewhat similar to the one in Dr. Cohen's case. Undoubtedly a spicule of bone penetrated the carotid artery in the cavernous sinus, causing an immediate pulsating exophthalmos. In the other case there was a spontaneous rupture, apparently of the internal carotid, in all probability from an aneurysm within the cavernous sinus. This spontaneous rupture occurred in the early morning, while the patient was asleep. She was a woman, aged about 58, with a negative Wassermann reaction. I should like to report that both of these patients were operated on by Dr. Eagleton and myself. We used the same procedure; namely, ligation of the internal carotid artery with a strip of fascia lata taken from the thigh of the patient. In the case of the woman, the vision before operation amounted to perception of fingers, there being countless hemorrhages all through the fundus, due to venous stasis. The subsequent course of the case was that the vision gradually returned to normal, the exophthalmos disappeared, and the head noises disappeared entirely without any untoward symptoms following the operative procedure. I saw the patient during the past year; her vision was still normal, and there was no indication of any unusual appearance about the eye.

In the traumatic case, the outcome was less fortunate. This man unfortunately had an old traumatic cataract on the eye that was not affected by the pulsating exophthalmos and the vision in that eye was nil. In the eye that had been affected by the injury, the vision still remained good, but in spite of ligating the internal carotid artery, then the common carotid artery on that side, and later on, the internal carotid artery on the other side, operations performed at intervals of from three to four months the patient was unable to close the eye satisfactorily, with the result that he developed a chronic corneal abscess. Even after the lids were sewn together and a paracentesis was performed, the man ultimately lost the vision in the eye. The first case, however, with a history of five years' duration of normal vision, is worthy of note.

BILATERAL RETINITIS PROLIFERANS. DR. GEORGE H. BELL.

Four months before presentation, a man, aged 34, was handling a bomb which exploded.

X-ray plates showed a number of metallic foreign bodies, chiefly in the infra-orbital regions. X-rays of both globes were negative, but there was a fractured malar bone. The patient was seen in my clinic at the New York Eye and Ear Infirmary for the first time on Nov. 9, 1928. The corneas were clear, and the tension was normal.

The picture of the fundi is as follows:

In the left eye, the vitreous is a little less solid than normal and contains many fine opacities. The optic nerve head is yellowish in color, and the margins are indistinct, due to the vitreous opacities and to postinflammatory changes present. The peripapillary region around the nerve head is blurred. Nasally, there is a triangular area bounded by two white lines at the angle of which is a bluish-gray spot deep in the choroid, the retinal vessels crossing over it. The triangular area is irregularly pigmented and probably represents a disturbance in the pigment epithelium; the white lines simulate a rupture of the choroid. Temporally, the

huge patch of connective tissue, which lies on an area of sclerosed choroid with secondary pigmentation, extends downward; it is joined by the nasal white line proceeding far into the periphery below. About 3 disk diameters away from the lower margin of the nerve head, a fairly large linear hemorrhage is seen on this connective tissue. A little below this two tiny droplike old hemorrhages are seen, which are anterior; these are in the posterior layers of the vitreous. The vessels are in fairly good condition except where the connective tissue covers them. The vision in the left eye is 10/200, unproved by correction.

The right eye presents a similar picture, but one which is far less extensive. There are no bluish-gray spots. The vision in this eye is 10/200, unproved by the refraction. The slip lamp examination is negative except for a moderately large number of brown deposits throughout the vitreous, indicating a hemorrhage into that body. There is a limitation of motion up and down and there is a fracture of the lower orbital margin. The primary lesion must have been produced below with hemorrhage into the vitreous and with rupture in the choroid around the nerve head.

The positive Wassermann reaction probably has no bearing in this case.

This is the most extensive case of retinitis proliferans that I have ever seen.

PIGMENT DEPOSITS IN CONTRACTION FURROWS OF THE IRIS. DR. ALGERNON B. REESE.

This report will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. ARNOLD KNAPP: During the past year I have seen a case with these contraction furrows which were deeply pigmented. This was the first case which I had seen in which this condition was so well marked. A man, aged 62, had glaucoma and syphilitic optic atrophy. The iris was blue, and there were three incomplete circles of contraction furrow; and these were not reddish, as in Dr. Reese's case, but had definite brown pigment. Looking at the patient with the slit-lamp, it was possible to see on the surface of the atrophic iris the peculiar dust of brown powder seen in glaucoma; this was concentrated in the contraction furrows to form the rings of pigment.

CENTRAL SCOTOMA IN STRABISMUS. DR. JOHN N. EVANS.

One hundred private patients with strabismus were selected. Of this number, thirty-four were monocular convergent strabismus in hyperopia; sixteen of these were selected as meeting the requirements. The requirements were particularly rigid to insure the elimination of all conditions giving rise to central scotomas from other causes and to rule out unreliable subjects.

Three studies were made. The first study demonstrated the scotoma and outlined its characteristics. The second showed the relation of the scotoma to the reduction of visual acuity found in these subjects. It incidentally set the visual (form) acuity isopters for the ⅓ and 2 degree zones (one and two minute objects). The third study showed the relation of the scotoma and reduced vision to the wandering movements encountered in these eyes.

The conclusions outlined were:

1. These amblyopic eyes showed some squint, though only of 1 or 2 degrees.

2. One hundred per cent of the subjects had a demonstrable absolute central scotoma.

3. A visual acuity of about 6/22 was typical of these amblyopic eyes.

4. There were others in the family with a similar condition.

5. Moderate degrees of compound hyperopia and astigmatism were typical of the subjects.

6. The left eye was most commonly affected.

7. Less than 20 per cent of all cases were accepted as demonstrating pure strabismus, in the sense here stipulated.

8. The reduced vision was satisfactorily explained by the scotoma.

9. The "wandering movements" were satisfactorily explained by the scotoma.

10. It is possible to relate other elements of the symptoms complex to the scotoma.

The numerous maps of the scotomas of both the accepted and unaccepted series were supported by three statistical tables.

DISCUSSION

DR. PETER: During the last three years, there has been no subject in our specialty that has interested me more than this subject of scotoma in squint, and it has been a great pleasure to read Dr. Evans' paper and to hear his presentation. I believe that few of us realize the prodigious amount of work which is entailed in the preparation of the paper which he presented tonight. You will recall that he had to exclude many cases in order to make the paper of real value. Whether we can absolutely agree with all the details of his conclusions makes little difference. In the main, I think I can agree with practically everything he has said. He has brought us definitely nearer to the solution of this important question. The subject has been discussed for years, as you know. We are in doubt as to whether this scotoma is really a congenital scotoma, or, as some of us believe, a scotoma from nonuse. This paper helps us toward a better solution of this interesting subject. I will discuss briefly some of the conclusions which Dr. Evans has reached, and will add some clinical observations to the discussion.

In his first conclusion — "squinting eye shows some squint, if it be but one or two degrees" — I would like to suggest that a squint of one or two degrees should properly fall under the head of esophoria.

He found an average of 6/22 in visual acuity. The average in the studies which I have made is 6/15.

In his third conclusion, he states that he always found other members of the family with the same condition. The hereditary influence in the scotoma of squint is interesting. I doubt whether we can prove that there is any hereditary influence in the scotoma. I have always felt that the hereditary phase in squint tended in two directions: First, patients inherit the refractive error of their parents; second, they inherit a weak or defective faculty which is the background of squint. Some of you may not agree with me on that particular point, but I personally am convinced that it is the dominating factor in squint. There are other factors which are important, but I cannot feel that the amblyopia is a determining factor in precipitating the squint.

As to the question of the refractive error, hyperopia with astigmatism undoubtedly is a dominant factor, second only to a defective faculty of fusion. However, I believe that unequal refractive errors are even more important, particularly in the amblyopia of the nonsquinters. Practically all the cases of this type of amblyopia which we find in nonsquinters show a high degree of difference in the refractive error of the two eyes.

In my own series of cases, the amblyopia was divided equally between the right and left eye. There was a difference of only one case.

Dr. Evans reports 20 per cent of true squints. My own figures are much higher. In the cases of amblyopia which I have studied, manifest squint was present in 8 per cent, while the nonsquinters and the squints which spontaneously recover make up the remaining 20 per cent.

The lowered vision is due to the scotoma.

As to the technic, there are several methods of studying these cases. I have not used the Schlösser method with red and green filters, but usually use the Ferree-Rand perimeter, the point of fixation being a very low filament of light. A study of the scotoma raises the question as to whether the condition is an absolute or a relative scotoma. I feel that it is a relative scotoma in most of the cases, because practically all the patients can definitely see this little filament of light in the center of the scotoma.

The scotomas which I have been able to chart have been just a little larger than those which Dr. Evans presented. They extend two or three degrees around the point of fixation, and their greatest extent is toward the blind spot of Marriotte. In some of these cases, I have observed the angioscotoma extending from the blind spot, but not definitely to the central blind spot. However, this may be because I have not employed Dr. Evans' technic.

The etiology is a subject on which I differ with Dr. Evans. Is it a congenital scotoma, as Heine believed, or is it a true amblyopia ex anopsia? There are three clinical groups of cases to be considered. The first group includes the cases of amblyopia with manifest concomitant squint. This group comprises 80 per cent of the cases. The second group should include cases of amblyopia with spontaneous recovery from squint; these usually occur in adolescents and in adults. The last group, numbering about 10 per cent of the cases, includes cases of amblyopia without squint. In this group, a few children 10 years of age or older are found, but most of the patients are adults.

Uhthoff says that the ex anopsia theory is the simplest. It explains the unilateral character of the condition, the improvement in a few cases, and especially recovery of vision after loss of the good eye has occurred; yet there are many facts which point to the theory of congenital amblyopia. It is easy to pick flaws in any theory. I do not propose to pick flaws, but believe it will be more constructive to give the reasons for my belief in the ex anopsia theory.

One of the chief points which tends to prove the theory of congenital amblyopia is a disinclination to believe that amblyopia can develop from nonuse. Three clinical groups furnish opposition to this thought. First, there are the cases of congenital cataract. It is agreed that congenital cataract should be operated on as early as possible. I could give many quotations from men of authority who insist on early operation because of the fear of the development of amblyopia from nonuse. Second, there is the group of cases of obstetric palsies. Gifford wrote on this question about four years ago. He gave the statistics of a great many cases, and his conclusions were that while many of these patients showed equal vision in both eyes, many of them had poorer vision in the squinting eye; in some cases the amblyopia was of high degree. In these cases of paralytic squint in the group with obstetric palsy, there is a reason why there should not be a uniform deterioration of vision in the squinting eye. Most of the patients have fusion when the eyes are turned in a certain direction away from the field of the paralyzed muscle. A certain number of these cases will necessarily retain their full visual acuity. In other instances, when the refractive error enters into the case, amblyopia may occur just as it does in concomitant squint. The third group consists of the cases which Claude Worth has so well presented in his book on squint, cases which I do not believe we can ignore. No one can read the review of the history of his

cases without noting that certain cases are definitely shown, by experimental methods, to have the squint and amblyopia transferred from one eye to the other. I do not use the treatment with atropine which he employed, which led to the discovery of those facts. Atropine was used for four or five months. During this period the patient failed to report. On the patient's return, the squint and the amblyopia were both found to be transferred to the other eye. That is incontrovertible evidence which should be accepted.

The other fact which leads some writers to espouse the theory of congenital amblyopia is their failure to improve the lowered vision which results from amblyopia. Uhthoff and Heine admitted that they were unable to improve the vision to any great extent. They therefore concluded that they probably were dealing with cases of congenital amblyopia, and not amblyopia from disuse. "By clinical evidence," Heine says, "we can hardly prove the amblyopia is congenital." On the other hand, he has no pathologic evidence to offer, nor does he find any experimental evidence to support his belief in congenital amblyopia.

Clinical evidence does tend to prove the theory of ex anopsia in squint. There are two facts on which I base my conclusions. First, there are isolated instances of amblyopic eyes which have recovered useful vision after loss of the fellow eye. Most of these have not been studied with any degree of care, but undoubtedly there are many in the group which are the result of amblyopia dating from childhood. I have had two of these patients brought to my attention recently, both of which, after losing the good eye, recovered good vision after fifty or sixty years of nonuse. There are isolated instances of this kind which tend to prove that these amblyopic eyes, dating from childhood, can be improved under certain circumstances. The chief facts on which I base my conclusion are the result of my own experience in the treatment of amblyopia. Some years ago I employed the same methods of training which all of us are using, and my results were rather uniform, but not satisfactory. Vision in the squinting eye improved to 6/12, but not often beyond 6/12. During the last three years, I have been paying special attention to this work, and have given these children rather intensive training. The results have been for the better. The slides which I had hoped to throw upon the screen were cases which I selected at random, most of them showing definite improvement after training. In these cases, the average correction vision by glasses is 6/15 before training. The average vision after training was a little better than 6/7. Four of these patients are still under training, and in time I am sure will be brought up to 6/6 or 6/5. If the patient is seen early enough, before 10 years of age, better still, before 6 or 7 years, I have found it possible to restore vision to nearly normal and to cause the scotomas to disappear.

The method of training is the last point in my discussion. My present method of training differs from that which I employed in former years. I feel that we are not insistent enough in the details of training. I do not use atropine, but I blindfold the good eye. That is done on an average of from one to six hours a day, depending on the degree of the amblyopia, and also on the age of the patient. I find that if the good eye is bandaged while the child plays around or is doing ordinary work, the results are not nearly so satisfactory as when the eyes are used at close range. The mother is instructed to interview the school teacher and ask that the good eye be covered for an hour and a half in the morning, and an hour and a half in the afternoon. The child at first rebels because fixation is not good. Practice at home with kindergarten methods, using rather large objects at first, and as the vision improves, smaller objects, prepares the child for school training in about a week. This method improves the vision rapidly. There are few of these children who cannot be trained to read type 0.37 on the chart. One

or two cases have failed to reach this degree of efficiency. I am not sure but that there are a few cases which might be called true congenital amblyopia, but the majority, in my experience, are the result of nonuse. That the vision can be restored is evidence of this fact. Distant vision is a little more difficult to restore. At present I am trying to evolve different methods of focusing vision on some definite object at a distance—first, 10 feet, and then 20 feet—and believe that there is a possibility of improving the distant vision of most of these patients. At the near point, it is rare to find a patient under 10 years of age who cannot be trained to read type 0.50 or 0.37.

DR. ALGER: A paper like that which Dr. Evans has just given raises so many points for discussion that one hardly knows where to begin. From time immemorial there have been patients whose eyes were normal enough to look at but had imperfect vision or perhaps no vision at all. The latter were grouped under the convenient term amaurosis, while those whose vision was merely dimmed were classified as amblyopic.

The invention of the ophthalmoscope revealed a variety of unsuspected lesions of media and fundus and gradually one disease after another was split off from the amaurotic group till now there is practically nothing left and the term is almost obsolete. The use of the word amblyopia has also been much narrowed by the discovery of lesions large enough to be visible with the ophthalmoscope. But there were many cases with apparently perfectly normal fundi which still had to be classed as amblyopic because our diagnostic skill did not permit of any better classification.

Then came the great improvement in subjective examination and particularly the refinements of field-taking which enabled the ophthalmologist to localize the trouble more or less exactly in brain, optic nerve or retina, until today, one rarely feel justified in making a diagnosis of amblyopia in an adult without some qualification as an essential part in its intelligent treatment.

Even in the case of young children one can exclude gross lesions of the fundus with the ophthalmoscope, estimate quite exactly the refractive error and in most cases get a good idea of the vision in each eye. If it is much reduced the causes for such a condition in an adult are sought. X-rays and Wassermann tests are made, and one searches for focal infections and the like.

But if the child has even the slightest sign of strabismus one is likely to abandon further investigation and fall back on the old diagnosis of amblyopia. This was originally a congenital condition for which nothing much could be done, except cosmetically.

Then came the theory of amblyopia ex anopsia which many physicians and most laymen interpret as meaning that a perfectly healthy eye which suppresses its images for a long time is likely not only to lose its central vision but to become practically useless. The logical treatment runs from continued bandaging or atropinization of the good eye to operations for strabismus in early infancy. Now amblyopia ex anopsia is almost a pure inference. It cannot be proved in a single case that the eye ever had a central vision which could be suppressed. One cannot exclude lesions of the fundus too small to be seen, or many conditions which are known to damage the central vision in adults and which might easily have occurred during the first five or six years of childhood, such as lesions of brain, nerve or retina. It cannot be explained why suppression should cause a macular dysfunction while all the rest of the retina which is just as much suppressed suffers no harm at all, or why a child with a barely perceptible squint or perhaps none at all should lose his central vision while another apparently much worse keeps his almost intact.

If by training and care the macular function of any considerable proportion of these children could regularly be restored, it would be a very strong argument for the theory. Of the thousands and thousands of children who have been watched and trained and operated on, however, the central vision of the squinting eye remains, for the most part, unimproved, while that of the totally neglected ones gets little if any worse.

There are to be sure almost miraculous restorations of function in a very few cases in which the better eye has been accidentally destroyed but I agree with Dr. Peter that these should be viewed with scientific distrust. Any case of this sort that is seen should be carefully studied and reported.

Field studies such as Dr. Evans has been making are, of course, absolutely impossible during early childhood when they would be the most valuable. But his observations correspond with those of others as to the presence of a pretty definite absolute central scotoma in almost all the cases if one has the patience and the skill and the machinery to bring it out. Heine thinks that they can practically all be proved congenital, and he attaches little importance to the hemorrhage from compression and neuritis which other observers have featured as the probable causes.

Dr. Evans' cases being selected because of the occurrence of other squinters in the family might more probably be illustrations of hereditary defects, whereas the scotomas due to hemorrhages would probably be larger and less uniform.

The commonest injuries at birth are multiple retinal hemorrhages with edema of the disk. If I remember correctly, there is a field chart in Dr. Peter's book which might follow such a condition.

But the hemorrhages of the new-born infant are said to clear up with marvelous rapidity and often perhaps, as in the adult, leave no trace in the field. It would be well worth while to keep notes of the final results in adults of the various types of hemorrhages over a period of years. My impression is that a hemorrhage at the macula is almost certain to destroy function, while peripheral hemorrhages often do not involve essential structures.

I must confess a great skepticism as to amblyopia ex anopsia but I would not for a moment be understood as arguing that there is no such thing. Quite possibly we may sometime be able to differentiate our cases much better than we do now and distinguish the hopeful cases from the hopeless. At present, however, the results seldom justify the time and the patience and the expense involved in orthoptic and fusion training.

DR. EVANS: You must remember that this is a study of only one small group. The difference between esophoria and esotropia comes up. Is not a squint a squint, whether it is of 1 or 30 degrees? As I understand esophoria, it is a tendency and not an existing condition under ordinary circumstances. It is perhaps a quibble to put it that way, but it is an excuse to account for the scotoma. As to amblyopia in nonsquinters; unless you can prove that they have not 1 or 2 degrees of squint, how can you say they are nonsquinters? A half a degree of squint is still a squint. Perhaps I quibble again.

I am sorry if I left the impression that I was using Schlösser's method. My object was white, not complementary to either filter. Probably the results might be the same. I mentioned that in the complete paper. I tried to point out that I did not think that my method exactly corresponded to that of Schlösser. Heine used a similar method, however. To use a small light as a test object, one must eliminate irradiation. The bulb of the ophthalmoscope itself is larger than the scotoma, so that the result of mapping a small scotoma would be a relative defect. Perhaps I am wrong. The theory of uncovering can account for the scotoma by

disuse and the congenital foundation; thus we do not have to distinguish between an amblyopia ex anopsia and a congenital amblyopia. I do not know what you mean by a congenital amblyopia. It needs more consideration. If an eye squints and has a vision of 1/200, and then the eye improves to 6/12, that does not mean that the eye has recovered from the amblyopia. It will only mean that the eye has learned the trick of fixation with a more central isopter. You have got to get a vision of 6/6 before you can say that the eye has improved or recovered from the amblyopia due to the presence of this scotoma. It is impossible to discuss the minute details of the problem because it means discussing the whole problem of strabismus.

In relation to Dr. Alger's remarks, it is impossible to rule out every little consideration, like a minute hemorrhage at birth. I did not try to specify whether I thought the cases were congenital or acquired, or what type they were. I simply specified that I was going to study a definite group and what constituted that group.

The bridge going from the central scotoma to the blind spot is not characteristic by any means of this scotoma. I think it has been pointed out that practically every central scotoma has a connecting bridge, but I cannot cite the source of the original observation. This bridge has the characteristics of the angioscotoma. That is why I do not think it is a defect of fiber bundles. It is not necessary to review all the angioscotomas in order to properly get that point.

PRERETINAL ARTERY. DR. ISADORE GOLDSTEIN and DR. DAVID WEXLER.

An anatomic study of preretinal artery was demonstrated. A full report will be published.

PERIPHERAL IRIDECTOMY IN CATARACT EXTRACTION. DR. EUGENE M. BLAKE.

This paper will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. BEN WITT KEY: I think we should be grateful to Dr. Blake for the presentation of this paper on peripheral iridectomy in the extraction of cataract which shows his thorough study of the subject. Furthermore, it is beautifully written and clearly stated. I confess I have never been an advocate of the peripheral iridectomy, and perhaps this is one reason why I have been asked to open the discussion on this paper — in order that the bitter may be mixed with the sweet and thus insure a broader view of the subject. Since hearing this paper, however, I shall certainly make the peripheral iridectomy in a number of cases for the purpose of a closer observation of it.

In the first place, I have always preferred the combined extraction in the great majority of cases, and believe that simple extraction is justified only in selected cases; that is, in mature cataract or sclerosed lens, with a deep anterior chamber, in patients less than 65 years of age. When iridectomy is advisable, I have been able to recognize no advantage of the peripheral iridectomy over the complete iridectomy, except in one point, which Tennent points out; namely, that it entails less risk of incarceration of tags of the lens capsule. I believe that when incarceration or plastering of the pillars of a coloboma in and near the wound takes place, or where iris is incarcerated after simple extraction, the same forces (accident, restlessness, an improper section, soft lens matter, pressure, etc.) would be just as apt to cause the same complications if peripheral iridectomy were performed.

I wish to congratulate Dr. Blake, however, on the views which he has expressed in his paper tonight. From now on, I shall make the peripheral iridectomy more frequently. Dr. Weil, Strasburg, recently performed his operation for me at the Infirmary. He used the lance knife for the corneal section and made a peripheral iridectomy. He is most dextrous and the result was all that could be desired.

DR. EDMUND SPAETH: It is rather interesting that I was first satisfied of the value of this procedure by Dr. W. H. Wilmer, when he recommended to me the same article to which Dr. Blake refers, that is, Pflüger's essay of 1897. The advice was given during a conversation which followed Barraquer's presentation of phaco-erisis, at the Washington session of the International Congress of Ophthalmology in 1922. A year later, I was sent with a letter of introduction to Professor Hess at Munich, but to my great regret and loss, he was then ill and died shortly after. I did, however, get his technic from his senior assistant at the Augen-klinik, Privatdozent Dr. Arnold Passow.

As Dr. Blake has stated, Professor Hess has, rather unfairly, received almost universal credit for this procedure, but this is due perhaps not so much for origination (this belongs to Pflüger) as for bringing the operation so positively before the ophthalmologic world by his large series of consecutive operations.

One might add, however, that of the several iris forceps recommended for this step in the operation, Dr. Dimmer of Vienna stated that "the Hess forceps was the only one which allowed one to do this peripheral iridectomy without danger from too wide an opening of the corneoscleral flap." In fact, he showed me that one could readily do the iridectomy with the Hess forceps and, with a double-blunt de Wecker pince scissors, wholly intra-ocularly.

The history of peripheral iridectomy is written in the history of the development of the modern operation for cataract. The conjunctival flap, the corneal or corneoconjunctival suture, and the peripheral iridectomy, are, as Hess once said, the triad of most importance in satisfactory results. As a matter of fact, these three steps, with a fourth, Hess covered most exhaustively in his Pathologie und Therapie des Linsensystems, while Professor of Ophthalmology at Würtzburg. This appeared in 1905, in the Graefe-Saemisch Handbuch der Gesamten Augenheilkunde. The fourth factor which he covered at that time concerned the intracapsular method of extraction of the lens. He discussed his reasons for and against intracapsular extraction. Some of these points, however, are foreign to the paper under discussion.

The stimulus behind its development is prolapse of the iris; delayed closure of the anterior chamber, the necessity for reoperations, and, too often, secondary glaucoma are the complications. I wonder if the two biggest factors which have prevented its more universal adoption are not, first, the preliminary iridectomy so often considered necessary in out patient clinical practice as well as in private practice; and second, the thought that this step in the operation is difficult. The latter point, Dr. Blake has answered rather cleverly. I am not defending the preliminary iridectomy; this is simply a statement which will be brought up again in a few moments.

If the danger of this complication, namely, prolapse of the iris, can be offset by either peripheral iridotomy or iridectomy, then it is possible to obtain a more nearly ideal result in postoperative cataract. This seems to be the problem.

There can be no doubt whatsoever that the round pupil, with a preserved mobile sphincter iridis, is ideal. Dr. Blake has dealt with this in his discussion of the advantages of the intact sphincter in extraction of the lens, when he quoted Tennent and Hudson. Vitreous prolapse is not as frequent, judging from the

literature; with a capsulotomy operation, capsule tags are less likely to become incarcerated in the wound and, a fact perhaps almost as important, hemorrhage from section of the iris cannot occur before extraction of the lens has been consummated. On the other hand, it is not uncommon to have more or less difficulty in extractio lentis with an intact sphincter, especially, as Hess pointed out, "when one is working with a large swollen lens or with a large hard nucleus. Also with the capsulotomy operation, it is not uncommon for large cortical masses to remain as an after cataract." This is a matter to be considered in recommending wide preoperative dilatation of the pupil before the operation.

The postoperative advantages of a round pupil are so evident, as Dr. Blake has shown, that every one must and seemingly, does agree to its advantages.

The peripheral iridectomy procedure cannot be applied to all cases, however. Dr. Blake, in mentioning some of the contraindications, quoted the complicated cases discussed by Pascheff. In addition to these combined extractions a certain number of indications for preliminary iridectomies are worth considering. Among them are the cases of immature cataract with swelling of the lens and secondary ocular hypertension; also those with the nuclear form of cataract, and cataract with high arterial hypertension in which a subretinal hemorrhage may occur, and in which it is either impossible or dangerous to reduce the arterial tension to a lower level. Even this does not change the state of the sclerosed walls of the vessel.

It is necessary to use the same discretion and judgment that is demanded for the operative treatment of the various types of glaucoma. In addition, it is quite possible that a complete iridectomy is necessary after the corneal section has been completed because of immediate prolapse of the iris and its stubborn resistance to reposition. This occurred several times while I watched Hudson work at Moorfields in London. The complete iridectomy was necessary.

As to the peripheral iridotomy, "its achievement is no more simple," states Lindner. "In fact, it is more difficult unless done before the extraction, it does not give the same easy access to fluids from the posterior to the anterior chambers, and it cannot be truly peripheral without an iridodialysis accompanying it." But that it does succeed, in spite of these facts, thus strengthening even more the position of those operators who practice the peripheral iridectomy, is shown by the series of Schweigger's one hundred cases presented in 1898. In this series he did an iridotomy parallel to the line of the limbus and as close to the root as was possible. The series had only two cases of complicating prolapse of the iris.

I think that we all would appreciate it if Dr. Blake would explain in more detail his procedure for dilating the pupil before the extraction, if he does this. Much depends on the immediate postoperative treatment. If atropine is used for dilatation before and after, this is less important. If eserine is used, however, after the operation, the limitation of its action by the atropine previously employed is not desirable. Dr. Blake used atropine following the operation. This also might be discussed in greater detail. In both the Lindner and Meller clinics in Vienna and in the Vogt clinic in Zurich, eserine is being used. Hess used it as well; Hudson, also, at Moorfields. Hess's technic was the use of 1 per cent eserine solution postoperatively and at the time of the first dressing. After the third day he changed to 2 or 4 per cent solutions of atropine.

Dr. Blake quoted Hudson in respect to this operation. It happens that I have sketches of his operative technic made at the time I was at the Royal Ophthalmic Hospital and I have brought these with me even though they are quite rough.

Dr. William McLean: I have done a great many peripheral iridectomies since I adopted the Barraquer method of phaco-erisis in 1922. In fact, the only

method of phaco-erisis in which I employ a full iridectomy to the pupillary margin is the kind in which I am unable to get sufficient mydriasis. At present, I am using atropine 1 per cent, instilled into the conjunctival sac one hour before the operation. The iridectomy is done as the second step of my operation for cataract, and there are two reasons why I do the iridectomy: First, I believe that the small opening of the peripheral iridectomy allows the entrance of air into the posterior chamber and facilitates the sliding of the lens out of its patellar fossa in the process of tumbling. Even with the pupil dilated, the margin of the iris rests against the periphery of the lens; thus, a closed chamber of the posterior space exists. It is to nullify this closed chamber that I employ the iridectomy and for that reason perform the iridectomy before the lens is extracted. Second, the peripheral iridectomy seems sufficient to prevent a prolapse of the iris after the completion of the operation.

I have had a great many good results with the peripheral iridectomy as an aid at the time of operation, and after recovery, the cosmetic effect of an aphakia with a round, mobile pupil is pleasing to most patients.

Dr. Eugene M. Blake: I put atropine in the eye in the morning, and operate in the afternoon, because I think it is much easier to get the lens out through a dilated pupil. Chandler says that in the cases in which he used eserine, he almost invariably had some posterior synechiae. Occasionally, atropine does offer a little complication. With the escape of the aqueous, the pupil contracts, even if it has been dilated. Occasionally, however, the pupil remains moderately dilated, making it more difficult to grasp the iris and perform an iridectomy. The last picture thrown on the screen was the Hess forceps, designed to be introduced into the wound, seizing the iris rather far down, near its attachment to the ciliary body.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION ON OPHTHALMOLOGY

Dec. 14, 1928

Cyril Walker, M.D., *President, in the Chair*

Retinal Hemorrhages. Mr. W. H. McMullen.

A girl, aged 16, had retinal hemorrhages, the causes for which could not be found. The blood pressure was normal, and physicians regarded the patient as normal. There was one hemorrhage on the disk, and another below it. Blurred sight occurred when a menstrual period was ten days overdue.

Embolism of a Branch of the Central Retinal Artery. Mr. W. H. McMullen.

In a case of embolism of a branch of the central retinal artery, the circulation was restored, but the patient had fairly frequent transient loss of sight in the other eye. She took a tablet of nitroglycerin when obscuration occurred.

Filtration of Aqueous After a Flap Sclerotomy. Sir Richard Cruise.

A man on whom flap sclerotomy had been performed was shown as an instance of the satisfactory termination of the operation for glaucoma. The scar was perfect. Aqueous was escaping from the anterior chamber, however, and the only

solution offered was that it was due to admixture of endothelial cells with ordinary scar tissue. There was no cause for anxiety when a case started to filter in this way.

Diffuse Corneal Opacities. Mr. Geoffrey Viner.

A married woman, aged 26, had diffuse corneal opacities. The slit-lamp did not reveal any inflammatory basis for the opacity. There were no changes in the cornea, no blood vessels and the opacity was homogeneous. Each eye had hypermetropia of 5 diopters, and, with correction, vision was 6/9. The condition was regarded as congenital.

Juvenile Tabetic Paralysis. Mr. Geoffrey Viner.

A man, aged 24, had juvenile tabetic paralysis; apparently, it was taboparesis of the juvenile type. In the six months before presentation, there had been a rapid failure of sight, due to optic atrophy. There was marked contraction of the visual fields. He had been treated for three months by malarial infection; vision did not improve.

Pituitary Tumor. Mr. Montague Hine.

A pituitary tumor was removed from a woman, with satisfactory results; the scar was not pronounced. Mr. Broster performed the operation. Vision increased from 6/36 to 6/9 in the left eye; alteration of vision did not occur in the right eye.

Hole in the Macula. Mr. Montague Hine.

In 1918, a man had an injury which resulted in a loss of central vision in the left eye. Peripheral vision was good. Nine years later, the patient developed optic neuritis in the right eye, the working eye, and was under observation at Moorfields for seven weeks. An x-ray picture did not reveal evidence of a skull fracture. The eye had a definite cupped disk. The exhibitor asked whether or not the condition could be related to the old injury.

Glaucoma. Mr. Montague Hine.

Glaucoma was present in the right eye of an old man, a similar condition, with hemorrhages, was present in the left eye. Also, under the right optic disk, there was a slate-colored oval patch, which seemed like a mole.

Albuminuric Retinitis. Mr. A. Caddy.

A girl, aged 14, had albuminuric retinitis and high blood pressure. On admission to the hospital, the blood pressure was 265. At present, it was 180.

Changes in the Cornea. Mr. T. Harrison Butler.

Examination with the slit-lamp revealed bright blue changes in the deep layers of the cornea. Irritation had always been present in the eye, and the patient had been treated for chronic conjunctivitis. There was no history of the entrance of a foreign body, a circumstance which might have offered some explanation of the condition.

Crystalline Deposit in the Cornea. Mr. C. Goulden.

A patient had an extensive crystalline deposit in the cornea, a sequel to prolonged inflammation. The patient came under observation in 1924, when a deep

keratitis in the left eye, rendered the cornea opaque. The Wassermann reaction was negative. When the patient was treated with atropine, the condition increased in severity. It was thought that ultraviolet light baths would be beneficial.

COPPER CATARACT. MR. C. GOULDEN.

Only one case with that rare condition, copper cataract, had been seen previously. A piece of metal entered the eye in 1916, and the sight was lost. The cornea was almost completely obscured by vesicles on its surface; the lens had practically disappeared, and the eye was blind. Examined in the direct path of reflected light, the lens gave the impression of a polished copper utensil.

RODENT ULCER. MR. A. D. GRIFFITH.

In a case of rodent ulcer, between the eye and nose, six applications of radium were given at weekly intervals, a total of 600 milligram-hours. In a fortnight after the last application, there was complete healing with a supple scar.

MUSTARD GAS BURNS OF THE EYE. MR. F. HECKFORD.

A late result of burning of the eyes with mustard gas was shown.. It was a case in which corneal ulceration occurred, ten years after the eyes had been burned with mustard gas.

PIGMENTARY CHANGE AT THE MACULA. MR. M. WHITING.

An unusual pigmentary change at the macula was shown in a woman, aged 64. It looked like melanoma of the ordinary type, except that the pigmentation seemed too dark. Five years previously, the patient had been struck in the eye by a piece of wood. There appeared to have been some hemorrhage, followed by the migration of pigment into that area. The defect of vision was due to a high degree of myopic astigmatism, which had remained uncorrected until shortly before presentation.

DEGENERATIVE CHANGES IN THE EYE. MR. M. WHITING.

A case of degenerative changes in both eyes was shown. The Wassermann reaction was positive, and the patient had undergone a vigorous course of mercury, potassium iodide and neoarsphenamine.

DISEASE OF THE CHOROID. MR. WILLIAMSON-NOBLE.

A case of disease of the choroid, accompanied by high blood pressure, was shown. On one occasion, the patient came down violently on one foot; after this, he noted that the vision was obscured.

Book Reviews

TUMORS ARISING FROM THE BLOOD VESSELS OF THE BRAIN: ANGIOMATOUS MALFORMATIONS AND HEMANGIOBLASTOMAS. By HARVEY CUSHING and PERCIVAL BAILEY. Price, $7.50. Pp. 219, with 159 illustrations. Springfield, Ill.: Charles C. Thomas, 1929.

For a quarter of a century, Cushing has acted as clinical diagnostician, surgeon, pathologist and follower-up of his patients with intracranial growths. These patients now number more than 1,520. By this means he has acquired unusual breadth of vision and correlated information which have greatly enriched his many writings on intracranial disease.

Meanwhile, observers—clinical, surgical and pathologic—have published numberless individual reports, each from his special point of view, which, while adding enormously to the bibliography, have not added considerably to the knowledge of the subject.

Furthermore, with his increasing experience and in the light of newer discoveries in this field, Cushing and his associates have studied and restudied his verified cases of tumor of the brain, and from this restudy with particular goals in view have come his books: The Pituitary Body and Its Disorders (with animal experimentation), 1912; Tumors of the Nervus Acusticus, 1917; Tumors of the Glioma Group, by Bailey and Cushing (a new classification of extraordinary prognostic significance), 1926, and the present volume, 1928.

There has been also a constant flow of shorter papers containing fresh observations and conclusions, many of which, I am proud to say, have appeared in this journal.

Cushing's classification of his verified intracranial tumors is as follows:

	Per Cent
Gliomas (varia)	42
Pituitary adenomas	19
Meningiomas	12*
Acoustic neuromas	9
Congenital tumors (varia)	6
Metastatic and invasive tumors	4
Granulomas	3
Sarcomas, papillomas and unclassifiable growths	4
Blood vessel tumors (of which the present book treats)	2

* (See Cushing and Eisenhardt: Meningiomas Arising from the Tuberculum Sellae, etc., Arch. Ophth. **1**:1 [Jan.] 1928.)

While the percentage of the last is small, many facts in regard to diagnosis and treatment are now made clear.

It may not be amiss to say a few words here to those who are not familiar with Cushing's book on the gliomas, which made up 42 per cent of his intracranial growths. After restudying the histology of his gliomas from an embryologic point of view, he devised a classification comprising more than a dozen varieties, and found that the lowly organized embryologic cell groups grow rapidly, are therefore malignant and soon cause death, while the most highly organized embryologic cell

groups grow slowly and are almost benign. Three of his patients in whom the glioma was incompletely removed lived, respectively, sixteen, eighteen and twenty-eight years after the onset of symptoms of disease of the brain.

The present volume treats of (1) venous varices, which are difficult to diagnose; (2) arterial aneurysmal varices in which arterial blood is forced into the veins with an audible bruit, both of which categories are regarded as malformed blood vessels rather than as neoplasms, and (3) types of cerebellar angiostomas, which are true neoplasms, composed of the elements that form primordial blood vessels.

The symptoms, which are more marked in the arterial than in the venous varices, are chiefly epilepsy, headache, brain palsies, homonymous hemianopic defects and deterioration of vision from papilledema.

The diagnosis of the second category may be aided by finding overvascularization of the head, a bruit over the lesion and a slight arterial coloring of the blood in the retinal veins.

As to treatment, decompression often relieves the symptoms, attempts at removing the vascular mass are disastrous and irradiation is of possible benefit. In a few cases, ligation of both common carotids has been of advantage.

A paper by Cushing and Bailey (Hemangioma of Cerebellum and Retina [Lindau's Disease] with Report of a Case, Arch. Ophth. **57**: 5 [Sept.] 1928) treats briefly of the subject of the third category in the present volume.

The symptoms of angioblastoma of the cerebellum do not differ from those of glioma; they are suboccipital tenderness, headache, vomiting, unsteadiness and papilledema. A concomitant angioma of the retina makes the diagnosis certain.

These benign tumors may be treated by decompression, or, without great danger, by extirpation or electrosurgical methods. When the tumor is cystic, the mural nodule from which the plasma transudes must be removed to prevent refilling.

We welcome this book by Cushing as we have welcomed his earlier books, and we look forward to further necromanic feats by him, after renewed study of his material with other goals in mind.

WARD A. HOLDEN.

DIE LABYRINTHREFLEXE AUF DIE AUGENMUSKELN NACH EINSEITIGER LABYRINTHEXTIRPATION NEBST EINER KURZEN ANGABE ÜBER DEN NERVENMECHANISMUS DER VESTIBULÄREN AUGENBEWEGUNGEN. By DR. R. LORENTE DE NÓ. Pp. 205, with 186 illustrations and 22 plates. Price, 15 marks. Berlin: Urban and Schwarzenberg, 1928.

In 1921, a capable pupil of Ramón y Cajal, Dr. R. Lorente de Nó, set out to study the anatomy and physiology of the pathways and centers of the vestibular apparatus. The present monograph is the result of his anatomic and experimental research which was begun in Spain and continued at Uppsala, under the bracing intellectual gaze of Professors Göthlin and Bárány.

With the acid of newly devised methods, he succeeded in recording at first the normal vestibular reflexes in each individual ocular muscle, then

the abnormal reflexes of the same muscles caused by extirpation of one labyrinth or by lesions artificially produced in the vestibular pathways and centers. To eliminate the visual stimuli and their possible bearing on the results of the examination, the vision in both eyes was destroyed beforehand.

His conclusions are definite and important. The quick phase of nystagmus depends on the integrity of the substantia reticularis. The stimuli necessary to produce the two phases of nystagmus (quick and slow) originate in the labyrinth, namely, in the same area for both phases, possibly in the crista. All the six muscles of each eyeball are involved in the phenomenon of labyrinth nystagmus. Any lesion of the substantia reticularis causes an abnormal reflex in the ocular muscles. There are no special centers for every vestibular reflex, and there is no single group of cells, or center, in the vestibular nucleus for each individual eye muscle.

The experiments were made on rabbits. It remains to be seen what will be the results of similar studies on higher animals and how far these studies can be reconciled with clinical and pathologic studies in human beings. The monograph is an important contribution to the subject of the neuro-anatomy and physiology of the vestibular apparatus, and careful study of its pages, drawings and tables is necessary for its comprehension.

MARK J. SCHOENBERG.

Directory of Ophthalmologic and Otolaryngologic Societies *

FOREIGN

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford, England. Time: July 4-5, 1929.

INTERNATIONAL

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

President: Dr. E. Treacher Collins, 17 Queen Anne St., London, England.
Secretary: Dr. E. Mark, Academisch Zickenhuis, Leyden, The Netherlands.
Place: Amsterdam, The Netherlands. Time: Sept. 5-13, 1929.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Portland, Ore. Time: July 10-12, 1929.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
President-Elect: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Atlantic City, N. J. Time: Oct. 21-25, 1929.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter R. Parker, David Whitney Bldg., Detroit.
Secretary: Dr. Emory Hill, 501 E. Franklin St., Richmond, Va.
Place: Hot Springs, Va. Time: June 10-12, 1929.

LOCAL

SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY OF THE LOS ANGELES COUNTY MEDICAL SOCIETY

President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SECTION ON OPHTHALMOLOGY AND OTO-LARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

Chairman: Dr. Walter A. Wells, 1606 20th St. N. W., Washington.
Secretary: Dr. J. N. Greear, Jr., 1740 M St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Lafayette Page, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. M. Earle Brown, Maison Blanche, New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital, New Orleans. Time: 8 p. m., third Thursday of each month from September to June.

SECTION ON OPHTHALMOLOGY, BALTIMORE MEDICAL SOCIETY

Chairman: Dr. Clyde A. Clapp, 513 N. Charles St., Baltimore.
Secretary: Dr. Aaron Robinson, 2335 Eutaw Pl., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St., Baltimore. Time: 8:30 p. m., fourth Thursday of each month from October to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Holbrook Lowell, 82 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.

THE KANSAS CITY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. T. S. Blakesley, Lathrop Bldg., Kansas City, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. William H. Luedde, Metropolitan Bldg., St. Louis.
Secretary: Dr. Leo L. Mayer, Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Donald Owen, Medical Arts Building, Omaha.
Secretary: Dr. Sanford R. Gifford, Medical Arts Building, Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7:00 p. m. Third Wednesday of each month from October to May.

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Lee W. Hughes, 1019 Broad St., Newark.
Secretary: Dr. Earle LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert M. Rogers, 680 St. John's Pl., Brooklyn.
Secretary: Dr. William F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave., Brooklyn.
Time: Third Thursday of February, April, May, October and December.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. M. S. Lord, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.

SECTION ON OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE

Chairman: Dr. B. Samuels, 57 W. 57th St., New York.
Secretary: Dr. T. B. Johnson, 30 W. 59th St., New York.
Time: 8:30 p. m., third Monday of every month.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Cecil B. Hert, 29 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

THE OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION OF THE CLEVELAND ACADEMY OF MEDICINE

Chairman: Dr. A. B. Brunner, Guardian Bldg., Cleveland.
Secretary: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Joseph L. McCool, 909 Stevens Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

THE PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.

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PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

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Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Place: Salt Lake City. Time: July 1-3, 1929.

SECTION ON OPHTHALMOLOGY, COLLEGE OF PHYSICIANS, PHILADELPHIA

Chairman: Dr. C. E. G. Shannon, 17th and Walnut Sts., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John G. McLaurin, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. M. Sellers, Medical Arts Bldg., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. P. Schenck, Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

EYE, EAR, NOSE AND THROAT SECTION OF THE HOUSTON ACADEMY OF MEDICINE

President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

THE SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. H. Roseborough, 302 Moore Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library, San Antonio, Texas. Time: 8 p. m., first Tuesday of each month from October to May.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. L. P. Allen, 145½ Main St., Oshkosh, Wis.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

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CONTENTS OF PREVIOUS NUMBER

APRIL, 1939. NUMBER 4

The Formation of the Aqueous Humor: Its Relation to Intra-Ocular and Vascular Pressures. Arthur M. Yudkin, M.D., New Haven, Conn.

Akinesis During Cataract Extraction. T. S. O'Brien, M.D., Iowa City.

The Intra-Ocular Tension Following Puncture of the Anterior Chamber. Peter Clemens Kronfeld, M.D., Vienna, Austria.

Embolism of Several Branches of the Arteria Ophthalmica. Ernest Waldstein, M.D., New York.

Ocular Hypotony in Retinal Detachment. M. N. Beigelman, M.D., Los Angeles.

The Etiology of Sympathetic Ophthalmia: Further Notes. S. R. Gifford, M.D., Omaha, and L. H. Lucie, M.D., Cheyenne, Wyo.

The Entoptic Phenomenon of the Blue Area: A Study of the Secondary Excitation in the Retina. Peter A. Snell, A.B., Rochester, N. Y.

Ring Abscess of the Cornea. S. R. Gifford, M.D., and C. E. Hunt, M.D., Omaha.

Ophthalmologic Review:
Ocular Residua and Sequelae of Epidemic Encephalitis. Aaron Barlow, M.D., Philadelphia.

Notes and News.

Obituary:
Lewis H. Taylor, M.D.

Abstracts from Current Literature.

Society Transactions:
New York Academy of Medicine, Section on Ophthalmology.
Royal Society of Medicine, London, Section on Ophthalmology.

Book Reviews.

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